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THE
LARYNGOSCOPE.

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RECURRENT OTOGENIC MENINGITIS.*†

J. H. MAXWELL, M.D.,
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Recurring attacks of meningitis associated with repeated episodes of otitis media presuppose some direct communication between the middle ear cavity and the subarachnoid space. Although this condition is relatively rare now, it was practically unknown before the advent of the chemotherapeutic and antibiotic agents. At that time the first attack was almost certain to be fatal.

The pathogenesis of this condition was outlined well by Dandy,[1] in 1944, when he was discussing cerebrospinal rhinorrhea and otorrhea. He stated that the most common causes of such spinal fluid leaks are skull fractures and operative injuries. Less common factors are erosion by tumors, or inflammation, and congenital malformations. This grouping serves well to define the abnormal pathways whether or not there has been a clinically demonstrable otorrhea. Certainly, skull fractures are much more frequently the cause of recurring otogenic meningitis than are congenital malformations.

Rowbotham,[2] in his text on "Acute Injuries of the Head," observes that in cases of compound petromastoid injuries, meningitis may develop during any stage of the healing proc-

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†From the Department of Otolaryngology, University of Michigan Hospital.

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ess. He states that delayed infection is more common than immediate meningitis and is due almost invariably to an intercurrent suppurative otitis media.

Spurling[3] comments on the serious implications of a traumatic cerebrospinal fluid leak from the ear, but states that, unlike those from the nose, a fistula into the ear nearly always heals within a week or two. This prompt healing has been noted by otologists after operative perforation of the dura during a mastoidectomy.

In an extensive study of skull fractures involving the ear, Grove[4,5] concluded that late meningitis complicating fractures through the petrous could be attributed to fracture into the otic capsule. It was the consensus of several authorities whose reports he reviewed that these transverse fractures through the otic capsule heal by fibrous rather than by bony union because of the endochondral bone involved. He was of the opinion that if meningitis did not develop soon after a longitudinal fracture, which usually does not traverse the otic capsule, and if an otitis media did not develop in the early stages of healing, the patient would be quite safe from late developing meningitis. He cited Fraser's discussion of Nager's[6] paper, however, in which there was reported a late developing case of meningitis after a longitudinal fracture of the petrous which involved the roof of the antrum but spared the labyrinth. He also reported one of his own cases in which meningitis developed six months after the fracture. Although there was no Roentgen or anatomical evidence that the labyrinth was not fractured, such was the assumption since the patient was not totally deaf in the involved ear. Grove reviewed reports of several observers who cited cases of meningitis developing three weeks to 16 years after fracture through the petrous. In these cases it was assumed that the late complication was associated with nonbony union of the fracture into the labyrinth.

It seems unnecessary to assume that late developing or recurring meningitis after a petrous fracture must depend upon a fracture of the labyrinth. It has been demonstrated

repeatedly that after fracture through the cribriform plate, a small sealed off vascular cuff of meninges, with or without brain tissue, may be trapped in the old fracture line and by means of its vascular connection provide the avenue of entrance of infection from the nose to the meninges. In an analogous situation in the petrous, it would seem quite possible for meningitis to develop late or to recur without involvement of the labyrinth in the fracture line. If the labyrinth is involved, it might be quite coincidental.

CASE REPORT.

J. R., No. 663690, a five-year-old boy, was admitted to University Hospital in May, 1949. He had suffered a skull fracture in March, 1948, on falling from a moving automobile. He had been unconscious for four or

Fig. 1. Case 1. Postoperative Roentgenogram. Note silver clip at the fracture line through the petrous.

five minutes and stuporous for the next 12 hours. Nine days later he developed signs of meningitis, which prompted the administration of penicillin, streptomycin and sulfadiazine for a period of one month. No

organisms were recovered from the spinal fluid during this episode. Five months later, a second attack of meningitis occurred, and pneumococci were recovered from the spinal fluid. The third attack, again due to a pneumococcal infection, developed in May of 1949 and was preceded by a left earache. After this attack he was examined at University Hospital where Roentgenograms failed to reveal definite evidence of a petrosal fracture. Also, there was no evidence of sinus disease or anterior cranial fossa fracture. The drum membranes were intact. There was a profound perceptive deafness but evidence of some residual hearing on the left side. Since surgical exploration did not seem to be indicated at that time, he was discharged from the hospital. He had another attack of meningitis in June of 1950 and was readmitted to the hospital in July. This time the X-ray films demonstrated a transverse fracture through the left petrous. An operation was done with the help of the neurosurgeons. On exposing the petrous ridge, the fracture line was demonstrated readily. The dura was trapped and attached firmly in the fracture line by scar. This was elevated and freed from the fracture line without evidence of opening the subarachnoid space. A strip of Gelfoam was placed over the fracture site after placement of a small silver clip for radiographic identification. The patient's recovery was uneventful and there has been no further trouble (see Fig. 1).

On rare occasions cerebrospinal otorrhea may be the result of a congenital malformation. Koch,[7] in 1950, commented on the great rarity of temporal bone meningoceles even though dehiscences in the tegmen of the tympanum or antrum had been noted on many occasions in cadaver material. He reported on the case of a 61-year-old woman who apparently had a meningocele near the sinodural angle. On admission to the hospital she gave a history of repeated bouts of headache, nausea, and vomiting associated with upper respiratory infections. The bulging left tympanic membrane was incised, allowing the escape of thin fluid. Meningitis developed in 12 hours and pneumococci type II-A were recovered from the aural discharge. After a few weeks, recovery was complete and the hearing was reported to be good. X-ray films demonstrated a pea-sized defect in the bone near the sinodural angle, the appearance of which did not suggest necrosis. It was concluded that the myringotomy knife had been inserted into a meningocele.

Koch also cited a report of Metz,[8] in 1941, describing bilateral meningoceles at the sinodural angles.

In 1951, Nenzelius[9] reviewed the literature on spontaneous cerebrospinal otorrhea and was able to find but two cases recorded. One of these was Koch's case mentioned above.

Nenzelius prepared a detailed report concerning a 15-month-old girl who had multiple congenital deformities. She was admitted to the hospital with acute meningitis and a bulging tympanic membrane. After myringotomy, there was a copious flow of clear fluid which continued for several days. She recovered promptly on antibiotic therapy, and the ear became dry. Six weeks later the meningitis recurred. On opening the pink and bulging drum membrane there was again a flow of clear fluid which was determined to be cerebrospinal fluid after chemical analysis. The fluid from the ear demonstrated a much lower white blood cell count than did the spinal fluid; also, Gram-positive diplococci were recovered on lumbar tap but no organisms could be grown from the aural discharge. A mastoidectomy was done, which revealed an abnormal otic capsule. The semicircular canals were absent, and there was no evidence of fenestra on the flat mesial wall of the tympanum. At the third operation, done because of recurring meningitis and the continued flow of cerebrospinal fluid through the nose by way of the Eustachian tube, the cavity of the deformed otic capsule was opened widely, then packed with a free muscle graft. This procedure resulted in complete healing.

Kline,[10] in 1933, reported a case of truly spontaneous cerebrospinal otorrhea. This occurred suddenly in an adult male while he was leaning over to lift an object from the floor. Death from pneumococcic meningitis occurred three weeks later. At necropsy a 2.5 mm. circular opening in bone and dura was discovered on the upper surface of the temporal bone between the arcuate eminence and the petrosquamous suture. A fistula leading into the brain was thought to be a duct in a congenital cyst because there was brain tissue in the fistulous tract.

CASE REPORT.

K. F., No. 720617. This five-year-old girl was admitted to University Hospital in January, 1952. She had had recurrent episodes of type IV pneumococcic meningitis at the ages of two, three and five years. A right earache preceded the last attack, which occurred in November, 1951. On examination, the child appeared to be in normal health. She could hear well in each ear, and although the right tympanic membrane seemed slightly more opaque than normal, there was no evidence of fluid

in the tympanum. Roentgenographic studies of the mastoids revealed a destructive process in the region of the right petrous apex which extended laterally across the floor of the middle cranial fossa and into the temporal squama (see Fig. 2).

Fig. 2A. Case 2. Roentgenogram demonstrating the destructive process in the region of the petrous apex and temporal squama.

On January 31, 1952, the floor of the middle fossa was exposed through a temporal approach by the neurosurgeons. A meningocele stalk was visualized as it penetrated the roof of the petrous and the floor of the middle fossa. This was connected to a large porencephalic cyst of the

temporal lobe. The petrous defect was packed with Gelfoam and covered with wax. The dural defect was too large to close. Recovery was prompt and the period since operation has been uneventful.

Fig. 2B. Case 2. Roentgenogram demonstrating the destructive process in the region of the petrous apex and temporal squama.

SUMMARY.

Two cases of recurring otogenic meningitis are reported.

The first of these resulted from a transverse fracture through the petrous. At operation it was found that the dura was trapped in the fracture line. It is concluded that this vascular meningeal cuff provided the pathway of infection from the ear to the subarachnoid space, and that any extension of the fracture line into the labyrinth was coincidental.

The second case was apparently due to the spread of infection from the middle ear to the stalk of a meningocele which penetrated a pneumatic cell in the petrous apex. There

had been no spontaneous otorrhea to suggest open communication between the middle ear and subarachnoid space. It can be assumed that the extension of infection took place by way of vascular channels.

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TRACHEAL AND ESOPHAGEAL COMPRESSION BY CONGENITAL VASCULAR ANOMALIES.*†

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and
ROLLIN A. DANIEL, JR., M.D. (by invitation),
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Malformations of the aortic arch and its branches which encircle the trachea and esophagus in such a way as to cause compression of these structures have interested anatomists for over a hundred years.[1-3] The clinical significance of these vascular anomalies and the importance of surgical correction in symptomatic patients have been emphasized in the last few years by the careful work of Gross, Neuhauser and their associates.[4-7] While in many cases partial or complete vascular "rings" are not associated with any symptoms whatsoever, these anomalies must be considered in the diagnostic evaluation of infants and young children presenting symptoms of tracheal or esophageal obstruction. A number of abnormalities may cause dysphagia or stridor in early life. These conditions include malformations of the larynx giving rise to so-called congenital laryngeal stridor, laryngeal and tracheal tumors, anomalous bands and webs, abscesses and tumors of the mediastinum and retropharyngeal area, laryngotracheobronchitis, foreign bodies, and congenital and acquired strictures of the esophagus and tracheobronchial tree.

The otolaryngologist is often responsible for the definitive diagnosis of the conditions listed above. Early recognition of congenital vascular anomalies which cause tracheal or esophageal obstruction is of great importance at the present time because of the availability of surgical relief.

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*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., Nashville, Tenn., Jan. 12, 1953.

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DEVELOPMENTAL ASPECTS.

The vascular anomalies which are most commonly associated with tracheal and esophageal compression are the result of abnormal development of the aortic arch system in early embryonic life. A review of the normal development of this system is helpful in understanding these anomalies: Between the fourth and seventh weeks of intrauterine life six pairs of aortic arches appear and partially encircle the primitive pharynx to connect the ventral aortic sac with the dorsal paired aortas. Caudal to the arches the dorsal paired aortas are fused at the aortic isthmus to form a single dorsal descending aorta (see Fig. 1). The first and second aortic

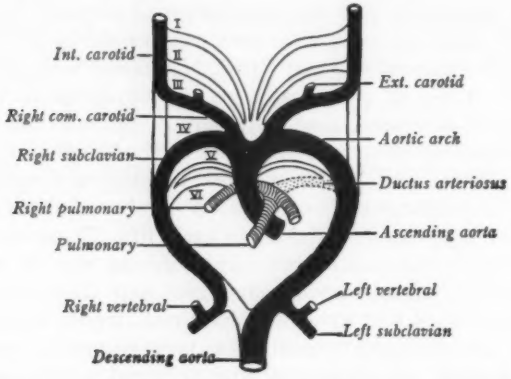

Fig. 1. Diagram of the normal development of human aortic arch and its branches from the embryonic aortic arches. (From Arey's Developmental Anatomy.[8])

arches quickly disappear, leaving the cephalic extremities of the dorsal aortas to form the definitive internal carotid arteries. The third arches persist as the permanent common carotids. The fourth left arch in continuity with the left dorsal aorta in the normal embryo forms the permanent arch of the aorta. The distal portion of the right dorsal aorta disappears. The fourth right arch in continuity with the proximal portion of the right dorsal aorta persists as the proximal segment of the permanent right subclavian artery; the right half of the ventral aortic sac adjoining this structure elon-

gates to become the innominate artery. The fifth arch disappears with no vestigial remnant, and from the sixth arch the ductus arteriosus and portions of the pulmonary arteries are developed. The permanent left subclavian artery is developed from the main stem of the sixth cervical intersegmental artery and its ventral ramus which arises from the left dorsal aorta beyond its junction with the fourth aortic arch. It migrates cephalad to its permanent position on the definitive aortic arch with the growth of the embryo.[8]

Vascular anomalies which produce obstruction of the esophagus or the trachea, or both, may be composed of any of several remnants of the primitive aortic arch system. Most commonly such malformations result from the persistence of those portions of the third and fourth arches and dorsal aortas which disappear in the development of the normal embryo and, less frequently, from the disappearance of those portions of the embryonic arches which persist where development is normal.

If the left fourth embryonic arch and the adjoining proximal part of the left dorsal aorta disappear and the right fourth arch and right dorsal aorta persist, a permanent *right aortic arch* results with the descending aorta coursing either to the right or the left of the spine. This condition may exist as an isolated anomaly or in association with dextrocardia, with or without situs inversus viscerum, and is usually asymptomatic. Compression of the trachea or esophagus may result in some patients with right aortic arch if *a.* the distal arch crosses transversely to the left behind the esophagus, or *b.* if there is an associated left posterior ligamentum arteriosum or patent ductus arteriosus which courses from the pulmonary artery to the left of the trachea and esophagus and thence behind these structures to join the distal portion of the right-sided aortic arch. A right aortic arch is encountered in about 20 per cent of patients with congenital cyanotic heart disease. In the great majority of these patients the aorta also descends on the right and an elongated ligamentum arteriosum passes from pulmonary artery to the left of the trachea to join the base of the left subclavian branch of the left-sided

innominate artery—in this situation there is no "ring" about the trachea and esophagus and in consequence no constriction of these structures.

If there is persistence of both right and left fourth arches in continuity with right and left dorsal aortas down to the point of fusion of the latter, a *double aortic arch* is formed

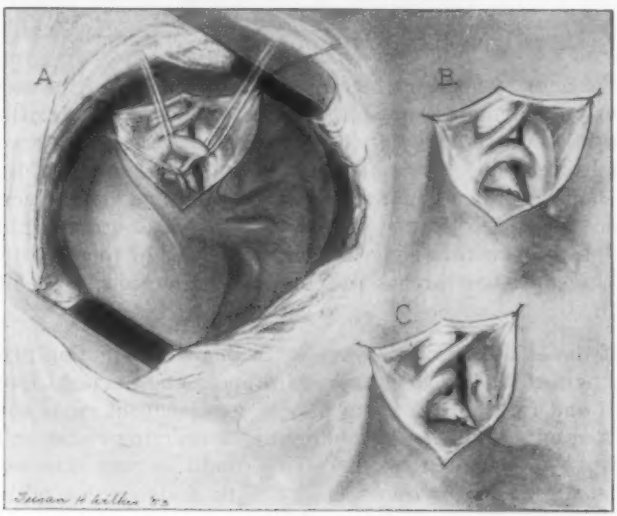

Fig. 2. Double aortic arch with left descending aorta (Case 1). A, B, C: Steps in relief of tracheo-esophageal compression by division of elongated patent ductus arteriosus and small anterior arch.

which is commonly the cause of disabling symptoms. In this situation the ascending aorta divides, the left branch passes anterior to the trachea, and the right branch usually passes behind the esophagus. The two arches then join to course distally as the left-sided descending aorta. Constriction of the trachea and esophagus usually is present with this abnormality. The two limbs of the aortic ring differ in size in most cases; the lumen of one limb is usually approximately the size of the normal aorta, while the other is much smaller (see Fig. 2). The left anterior arch is the smaller limb in the majority of instances.

Occasionally the lumen of the smaller of the two limbs of the aortic ring is obliterated in part, and that portion of the embryonic arch and dorsal aorta may be represented by a fibrous band or cord (see Fig. 3). While the descending aorta is on the left in most instances of double aortic arch, it is occasionally located to the right of the spine as in the case illustrated by this same figure.

Fig. 3. A: Double aortic arch with fibrous bands running behind esophagus to join distal end of smaller anterior arch to right descending aorta (Case 3). B: Release of tracheoesophageal compression by division of fibrous connection between arches and ligamentum arteriosum.

When the proximal portion of the right dorsal aorta which is in continuity with the right fourth arch becomes obliterated, rather than the distal portion, the right subclavian artery arises from the descending portion of the definitive aortic arch behind or distal to the origin of the left subclavian and usually passes obliquely across the mediastinum behind the esophagus to the right arm (see Fig. 4). While the retroesophageal position is that most commonly occupied by an aberrant subclavian, the vessel may occasionally cross between the trachea and esophagus and, rarely, in front of the trachea.[9] The mirror image of this condition occurs with a right aortic arch and a retroesophageal left subclavian artery. Aberrant subclavian arteries are common anomalies and in most instances are unaccompanied by symptoms; however, if

the aberrant subclavian is large in caliber and lacking in redundancy in its intrathoracic course, not only the esophagus but also the trachea may be compressed between the retroesophageal artery and the normally placed anterior aortic arch.

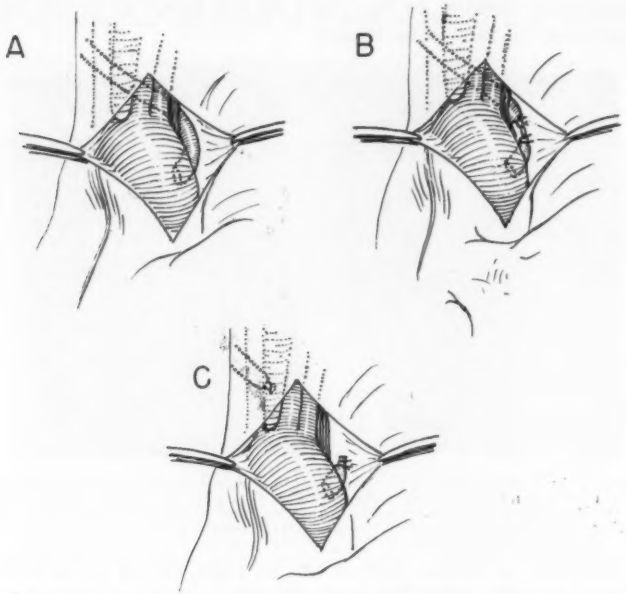

Fig. 4. Aberrant (retroesophageal) right subclavian artery (Case 4). A, B, C: Dissection, ligation and division of retroesophageal subclavian which in this case caused compression of trachea as well as esophagus.

Many variations of these vascular abnormalities have been described. Gross and Neuhauser have encountered several instances in which tracheal compression resulted from an anomalous innominate artery and others in which an anomalous left common carotid artery was responsible for obstructive symptoms.[7]

CLINICAL MANIFESTATIONS, DIAGNOSIS AND TREATMENT.

Despite the wide variety of anatomical arrangements assumed by these anomalies, their clinical manifestations fall

into a rather consistent pattern. Unless they are accompanied by other developmental defects in the cardiovascular system, abnormalities of the embryonic aortic arches are not associated with cardiac symptoms. Their primary effects are to produce mechanical partial obstruction of the trachea or esophagus, or both. Clinical manifestations usually become apparent during infancy and these are illustrated by the cases reported in brief below. In the majority of instances there is difficulty in swallowing (dysphagia lusoria) which varies in degree. This symptom may not appear until solid food is added to the diet of the infant. With more severe esophageal compression swallowing difficulties may present in the neonatal period. There may be hesitancy and regurgitation requiring repeated small feedings for maintenance of nutrition.

When tracheal obstruction is present, as is the case in most symptomatic vascular "rings," symptoms may be alarming. There is usually a "crowing type" of respiration which is associated with movement of the ala nasae, retraction of the lower ribs and sternum on inspiration, increase in the respiratory rate in some instances and auscultatory evidences of moisture in the air passages. Cyanosis may be present and if the tracheal compression is marked or pneumonitis becomes widespread, death of the infant is likely. Aspiration of secretions from the pharynx and even from the trachea fails to relieve the symptoms or to rid the patient of the rattling stridor characteristic of lower tracheal obstruction. The infant often has a tendency to lie with the neck hyperextended, breathing being somewhat easier with the trachea elongated and tensed. Frequently respiratory symptoms are exacerbated at the time of feedings.

Examination of the larynx of these infants will rule out congenital malformations and acute infections. The diagnosis can be confirmed in most cases by careful fluoroscopic and radiographic examination of the esophagus.[6] Moderately thick barium sulfate is the most satisfactory contrast medium. An indentation of the posterior esophageal wall at about the level of the aortic arch is confirmatory evidence of an anomalous

and obstructing vessel (see Fig. 5). Where a large aortic arch crosses the midline behind the esophagus, the forward

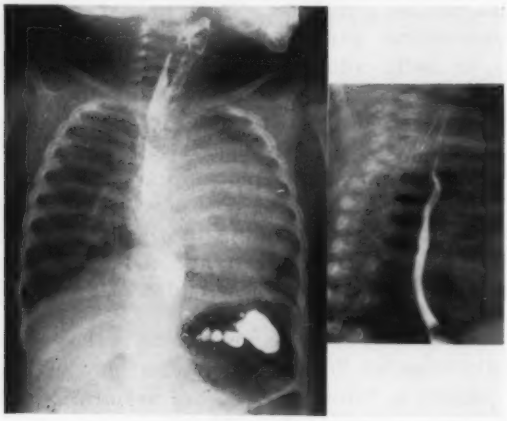

Fig. 5. Aberrant right subclavian artery (Case 4): AP and lateral films of chest with barium in esophagus.

displacement of the esophagus will be pronounced (see Figs. 6 and 7) and pulsation of the barium-filled esophagus may be observed fluoroscopically.

Rarely, tracheal obstruction may be severe without significant compression of the esophagus; in which case there is usually no artery crossing the posterior mediastinum. Where a vascular anomaly is suspected but anterior esophageal displacement cannot be demonstrated, a tracheogram, using iodized oil, should be performed. This is indicated whether the aortic arch is shown to be to the right or left of the trachea and esophagus. Confirmatory evidence of the vascular anomaly will be presented by demonstrable tracheal narrowing at the approximate level of the aortic arch (*i.e.*, near the lower end of the trachea).

Surgical treatment is based on relief of tracheal and esophageal obstruction by appropriate mobilization and usually division of the constricting elements of the vascular anomaly. Gross[7] has demonstrated the value of the routine use of a left

anterior thoracotomy in approaching these anomalies and has emphasized the importance of meticulous mediastinal dissec-

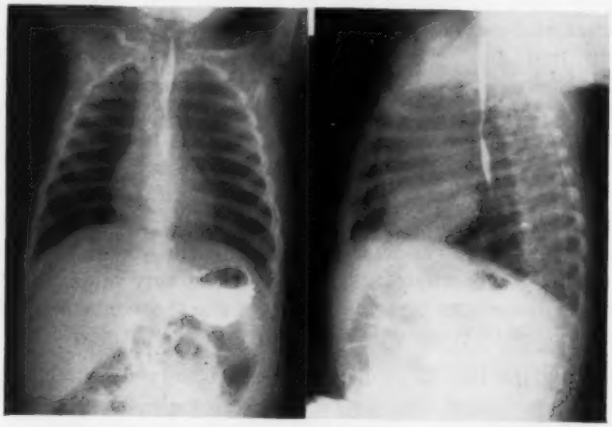

Fig. 6. Double aortic arch with left descending aorta (Case 1): AP and lateral films of chest with barium in esophagus.

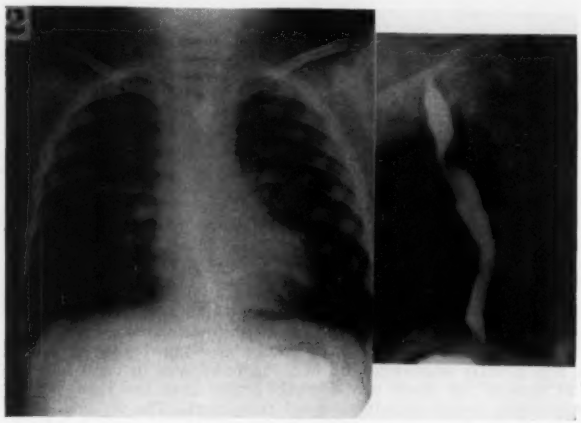

Fig. 7. Double aortic arch with left descending aorta (Case 2): AP and lateral films of chest with barium in esophagus.

tion with careful exploration of the anatomical relations of the entire aortic arch and all the great arterial trunks insofar

as possible in order to evaluate each anomaly accurately. With double aortic arch it is almost always advisable to divide the smaller arch in such a way as to break the ring about the trachea and esophagus with minimal disturbance of the major aortic flow and with particular care not to jeopardize the carotid circulation. An aberrant subclavian artery can always be divided with impunity. The ligamentum arteriosum should be divided as a routine in any case in which there is the slightest possibility that it contributes to the constrictive elements of the anomaly. It is important to mobilize the anomalous arterial structures widely and to divide all accompanying fibrous strands so as to permit the anomalous vessel to retract freely when interrupted. Great care must be taken in these dissections to avoid injury to the thoracic duct and to the important nerves and vessels of the mediastinum.

During the last 12 months we have observed four children with anomalies of the aortic arch which produced markedly disabling symptoms. Double aortic arch was found at operation in three of these patients. In the fourth an anomalous right subclavian artery, which gave rise to both esophageal and tracheal obstruction was found. The following case reports are illustrative of the anomalies most frequently encountered.

CASE REPORTS.

Case 1: P. G. F., a three and one-half months old white female infant, was admitted to the Vanderbilt University Hospital on Dec. 13, 1951, with a history of noisy respiration since birth. The stridor was constant, and it had been noted that breathing was made easier by placing the baby in bed on her right side and especially with the neck hyperextended. Respiratory stridor became much worse on crying, when she often became cyanotic. No difficulty in swallowing had been noted.

Examination revealed the infant to be well developed and nourished, alert and responsive. Significant physical findings were limited to the respiratory system. There was a loud "crowing" type of respiration, which was greatly accentuated when the baby was in the erect position. The left side of the chest was slightly larger than the right. There was rather marked inspiratory intercostal retraction. The heart sounds were normal, and the lungs were clear to auscultation except for loud transmitted stridulous sound heard best over the sternum.

Direct examination of the larynx revealed no abnormalities.

Fluoroscopic and radiographic examination of the thorax revealed marked posterior indentation and forward displacement of the barium

filled esophagus at about the normal position of the aortic arch (see Fig. 6). There was also evidence of narrowing of the lower portion of the trachea.

On Dec. 18, 1951, operation was carried out through a left transpleural approach. A double aortic arch was found, the left arch being smaller than the right, which crossed the mediastinum from left to right between the esophagus and spine. Marked constriction of the esophagus and trachea was demonstrated (see Fig. 2). A small ductus arteriosus, which appeared to be patent, extended from the distal portion of the left arch to the pulmonary artery. The ductus and the left arch were divided between silk ligatures. When this was accomplished there was marked retraction of the ends of the vessels, and the child's respiration, which had been stertorous, was greatly improved.

Following operation, the infant's course was greatly and dramatically improved. She was discharged from the hospital on the sixth postoperative day and has developed normally without recurrence of symptoms since that time.

Case 2: J. L. P., a white female, aged two years and three months, was admitted to the Vanderbilt University Hospital on March 4, 1952. At the age of three weeks it was noted that she had a wheezing type of respiration, thought to be caused by a respiratory infection. Following that

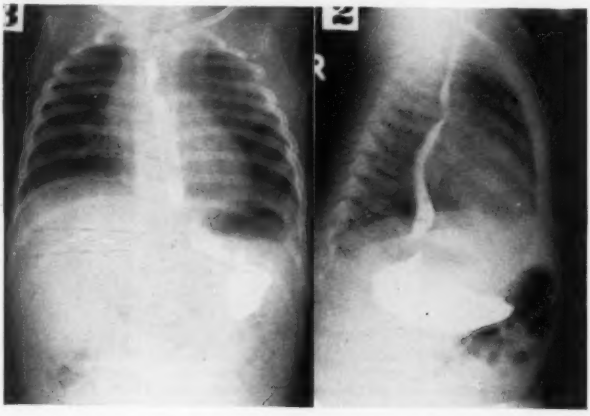

Fig. 8. Double aortic arch with right descending aorta (Case 3): AP and lateral films of chest with barium in esophagus.

episode there was frequent recurrence of respiratory infections associated with a wheezing and crowing type of respiration and often with paroxysms of violent coughing. The attacks became gradually more frequent and more severe and during the three months prior to admission a diagnosis of bronchopneumonia was made twice. She had had no apparent difficulty in eating, although she often refused to swallow food and fluids during the febrile illnesses.

Examination revealed her to be normally developed, well nourished and alert. There was wheezing and crowing with respiration, audible throughout the patient's room. Numerous expiratory and inspiratory wheezes were heard over the entire thorax. Examination of the heart revealed no abnormalities, and the remainder of the examination was not remarkable. There was no discernible cyanosis.

Studies of the blood and urine revealed no abnormalities.

Laryngoscopy, performed by Dr. William Kennon, revealed normal findings. The trachea was visualized and was thought to be narrowed by pressure upon its posterior wall by an extrinsic mass.

Roentgenographic examination of the esophagus with thick barium sulfate showed displacement of the esophagus forward and toward the left at the level of the third thoracic vertebra (see Fig. 7).

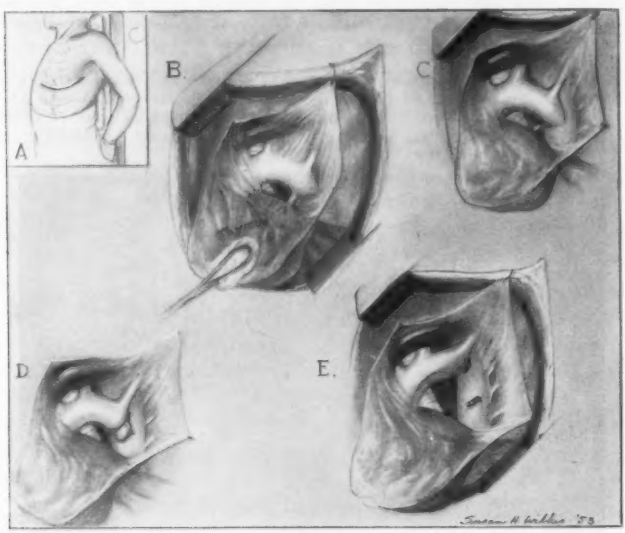

Fig. 9. Double aortic arch with left descending aorta (Case 2). A, B, C, D, E: Sequence of surgical steps in relief of tracheal and esophageal obstruction caused by this anomaly.

A diagnosis of congenital vascular anomaly was made, and operation was carried out on March 18, 1952. The left pleural cavity was entered through the left third intercostal space anterolaterally. Careful dissection of the mediastinum revealed a large aortic arch which extended to the right of the trachea and esophagus with the descending aorta slightly to the right of the spine. A smaller arch extended from the ascending aorta across the anterior aspect of the trachea and around the left side of the trachea and esophagus to join the descending limb of the right arch behind the esophagus (see Fig. 9). The common carotid and left sub-

clavian arteries arose from the smaller arch and just proximal to the origin of the left subclavian artery a very small patent ductus arteriosus extended from the smaller left arch to join the pulmonary artery. The ductus arteriosus was divided, and the left arch was then divided between the origin of the left subclavian and its junction with right arch posterior to the esophagus. The ends of the vessels retracted markedly and the child's respiration, which had been stertorous, was considerably improved.

Following operation the patient's respiratory wheeze, though considerably less marked, persisted even until the time of discharge from the hospital on the eleventh postoperative day. Thereafter, she slowly improved and at the present time has no objective evidence of tracheal or esophageal obstruction.

Case 3: R. E. S., a six months old white male infant, was admitted to the pediatric service of the Vanderbilt University Hospital on March 12, 1952, because of "wheezing" and repeated respiratory infections of four months' duration. The mother's pregnancy had been uneventful; the baby was the product of a full term normal delivery; weight eight and one-half pounds. Except for transitory cyanosis and respiratory distress for three or four hours after birth the neonatal period was uneventful. At the age of two months he developed the first of a series of severe respiratory infections, characterized by "wheezing," respiratory distress, cough and high temperature. A diagnosis of asthma was first made by his attending physician, but treatment with antiasthmatic medications was ineffective. Noisy, "crowing" respirations persisted, and it was noted that feedings were taken very slowly with frequent pauses. Because of persistence of these symptoms he was referred to this hospital.

Physical examination on admission showed a well developed and nourished white male infant with labored, rapid respirations accompanied by intercostal retraction and loud inspiratory stridor. There were diffuse rhonchi in both lungs with harsh breath sounds and both inspiratory and expiratory groans. Heart was not enlarged and there were no murmurs. The remainder of the examination showed no abnormal findings.

Routine laboratory examinations of blood and urine were normal.

Fluoroscopic and radiographic examination of the chest and the barium filled esophagus revealed a normal cardiac silhouette with a right aortic arch, and an indentation of the left and posterior aspects of the esophagus at the level of the aortic arch (see Fig. 8). A retrograde thoracic aortogram was technically unsuccessful.

Thoracotomy was carried out on March 17, 1952, under intratracheal nitrous oxide-oxygen and ether anesthesia. The chest was entered in the third left intercostal space and mediastinal dissection revealed the anomaly seen in Fig. 3. There was a right aortic arch with right descending aorta and a smaller left anterior arch from which arose the left common carotid and left subclavian arteries. Near the base of the left subclavian the left arch was drawn posteriorly and medially by fibrous bands which passed behind the esophagus, presumably to join the descending aorta. The ligamentum arteriosum arose from the small left arch at the base of the left subclavian artery and drew the pulmonary artery back against the trachea and left main bronchus. There was no patent vascular connection between the left arch and the right-sided descending aorta. After dividing the fibrous bands, which joined the left arch and the descending aorta, and the ligamentum arteriosum, the pulmonary artery and the left arch retracted anteriorly with prompt improvement in the ease of pulmonary inflation.

The infant was placed in humidified oxygen after operation and was given antibiotics in full dosage. Respirations were less labored and there was gradual diminution of stridor and rhonchi. Wound healed per primam. He was discharged on the thirteenth day after operation. His subsequent course has been one of gradual but steady improvement. Some stridor accompanied crying for two or three months and then disappeared. There has been no evidence of residual tracheal or esophageal compression for the last six months.

Case 4: J. S., a 14 months old white female, was admitted to the pediatric service of the Vanderbilt University Hospital on July 13, 1952, because of "wheezing" since the age of three weeks. The mother's pregnancy was uneventful and the infant was born at full term weighing six and one-half pounds. In the neonatal period she nursed poorly and gained slowly. "Wheezing" respirations appeared at three weeks and were made worse by feedings. Repeated severe respiratory infections necessitated frequent hospitalizations in other institutions. At seven months of age she developed severe bronchopneumonia, collapse of the right upper lobe and transitory cardiac failure. Atelectasis persisted for several weeks despite intensive therapy. Stridor, dysphagia and frequent bouts of pneumonitis continued up to the time of referral to this hospital.

Admission examination revealed an undersized, thin, pale baby with labored stridorous respirations. There was no cyanosis or clubbing. Breath sounds were harsh with scattered rhonchi in both lungs and inspiratory wheezes and groans. The heart was moderately enlarged with a systolic thrill and accompanying murmur over the base. The remainder of the examination was not remarkable.

Routine laboratory work showed a moderate leucocytosis and was otherwise within normal limits.

Fluoroscopic and radiographic examination of the chest and heart with barium studies of the esophagus showed cardiac enlargement, left aortic arch, increased bronchovascular markings, and indentation of the posterior aspect of the esophagus (see Fig. 5).

Bronchoscopy revealed larynx and upper trachea to be normal. There was severe narrowing of the trachea just above carina due to extrinsic pressure on posterior wall.

Diagnoses were congenital heart disease, acyanotic type (? interventricular septal defect), and tracheoesophageal obstruction due to vascular "ring."

Because of continued fever and signs of pneumonitis prolonged preparation for operation was necessary. This included penicillin and streptomycin in full dosage, oxygen, humidity and multiple small transfusions.

Operation was carried out on July 30, 1952, under intratracheal ether anesthesia. The chest was entered anteriorly in the third left intercostal space. Mediastinal dissection revealed a normal left aortic arch and an aberrant right subclavian artery which arose from the posterior aspect of the aortic arch at the same level as the origin of the left subclavian. This vessel passed behind the esophagus to the right arm and snugly compressed the esophagus and trachea against the aortic arch. The retroesophageal artery was doubly ligated and divided (see Fig. 4). As the chest was closed sudden cardiac arrest occurred. The chest was reopened immediately and cardiac massage begun at once. There was no response to vigorous resuscitative efforts.

Autopsy confirmed operative findings and in addition disclosed a high interventricular septal defect and chronic interstitial pneumonitis.

COMMENT.

Vascular anomalies which result from abnormalities in the development of the aortic arch and its branches are much more common than indicated by the reported incidence prior to the publication of the important observations of Gross and Neuhauser in 1946.[4-6] In many instances no physiologic impairment and no symptoms are produced by these anomalies. Tracheal and esophageal obstruction may result, however, and death of the patient may occur from sudden occlusion of the already narrowed trachea or from obstructive bronchitis and pneumonitis if the examining physician is not aware of the frequency of vascular anomalies which cause obstruction. Tracheal compression by a vascular "ring" may be erroneously diagnosed as congenital laryngeal stridor or, in the event of a superimposed pulmonary infection, as a simple laryngotracheobronchitis. Careful direct examination of the larynx and Roentgen examination of the barium filled esophagus should be carried out on all infants and children who have persistent or frequently recurring symptoms suggestive of tracheal or esophageal obstruction. These examinations should be carried out without general anesthesia because of the greatly increased anesthetic risk imposed by the presence of respiratory obstruction. The importance of antibiotics, humidity, suction and other measures to reduce infection and excessive bronchial secretions before operation cannot be overemphasized.

Following operation for the correction of obstruction caused by the pressure or constriction of anomalous vessels, relief of symptoms may be instantaneous and dramatic; however, in some cases a wheezing type of respiration may persist for days or even for several weeks, usually with progressive improvement and the ultimate disappearance of all disability. This is probably due to pressure deformity of the tracheal cartilage which tends to disappear after removal of the source of extrinsic compression and with the continued growth of the child.

Complete relief from disabling symptoms might not occur in patients who have other associated cardiovascular anomalies, such as pulmonic stenosis. The great majority of reported cases of vascular "rings" with obstructive symptoms, however, have not been associated with other significant cardiac abnormalities.

SUMMARY.

Congenital vascular anomalies are important causes of tracheal and esophageal compression in early life. The embryologic background, clinical manifestations, diagnosis and treatment of these anomalies are discussed. Several illustrative cases are reported in detail.

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TREATMENT OF ATELECTASIS IN THE NEWBORN BY STERNAL TRACTION.

A PRELIMINARY REPORT.*†

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The incidence of atelectasis in the newborn is very low, yet it is of major importance as a cause of death and is several times more frequent in the premature than in the full term infant. Clerf[1] states that atelectasis occurred 157 times in 13,515 live births. Ninety-five cases were in premature infants, while the remaining 62 cases were in full term babies. Herbert C. Miller,[2] in a review of fetal and neonatal deaths in 4,117 consecutive births, states that hyaline-like material in the lungs was the most frequent significant finding as a cause of neonatal death. Labate[3] gives "anoxia" as the most frequent cause of neonatal death.

The term atelectasis literally means "failure to expand" or "imperfect expansion." In common usage it covers three types of pulmonary collapse which occur in the human infant: *1.* that due to the resorption of air from a segment of pulmonary tissue distal to an obstructed bronchus or bronchiole; *2.* that due simply to failure of the pulmonary tissue to expand; *3.* that due to pressure changes exerted at the surface of the lung.

The aspiration type is easily understood. The laryngeal closure reflex is often poorly developed, especially in premature infants, so that the aspiration of gastric contents, formula, etc., occurs readily. This type of atelectasis responds readily to bronchoscopic aspiration, which is quickly and easily carried out even in the smallest infants.

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Farber and Sweet[4] and others have noted the presence of large numbers of cornified epithelial cells and fat droplets in the alveolar spaces and ducts of the lungs of infants dying before or shortly after birth. This, they believe, is the result of respiration attempted *in utero* by the asphyxiated infant.

Any consideration of the "failure to expand" type of atelectasis must take into account the changes that occur in respiration and, more particularly, the expansion of the lungs at birth. There is much experimental evidence that the fetus *in utero* does exhibit primitive respiratory movements; however, Clement Smith[5] cautions: "Present knowledge concerning the development of thoracic movements *in utero* does not allow the assumption that the onset of breathing at human birth is merely a continuation of a constant fetal activity." Two factors, according to Smith, acting on the central nervous system initiate the rhythmic respiratory cycle; the externally applied stimulus brought about by contact with the new environment, and the chemical stimulus of hypoxia produced by separation from the maternal circulation. Modifying this picture is, of course, the state of the central nervous system. Thus one sees the gasping, irregular type of respiration more frequently in the premature infant. According to Smith, high concentrations of oxygen rather than carbon dioxide tend to restore the higher centers and thus rhythmic respiration.

Farber and Wilson[6] stated that an infant's first breath is its most difficult one. They found that it takes pressure equal to approximately 25 cm. of water to expand the atelectatic infant lung. Smith states that a full term infant can exert pressure equal to 40 to 50 cm. of water on his lung, and sets this as the upper limits of safety. He advises a pressure of no greater than 25 cm. of water in an infant in respiratory distress for fear of rupturing portions of the lung already on the stretch.

The preponderance of evidence suggests that the initial expansion of the lung is an uneven, gradual process requiring hours, or, in some cases, days to complete.

Hilding[7] reports that Roentgenograms on the chests of 118 normal newborns showed one-third to have persistent atelectasis up to 24 hours. This is consistent with experimental work that he, as well as Wilson and Farber, did.

Clement Smith states that the diaphragm does most of the work in "pulling" the lung open. Hilding believes that the change from the "curled up" to the opisthotonic stretched-out position exerts a piston-like pull that opens the lung initially.

There is still difference of opinion regarding the presence or absence of amniotic fluid in the fetal respiratory tract. Labate states that it probably fills the trachea but not the alveoli. Gruenwald[8] found that fluid would expand the atelectatic infant lung with far less pressure than gas. This latter fact would seem to offer a logical explanation of its presence.

Bronchoscopy is a useful but often neglected procedure in these cases. It is without harm and is easily performed by the experienced. This is especially true with the advent of smaller instruments affording greater visibility[9].* It is not within the scope of this paper to discuss the details of infant bronchoscopy; if utilized promptly, however, even in the absence of bronchial obstruction, it can be life-saving, as the following case illustrates:

A male infant, born alive and weighing six pounds, showed cyanosis of the extremities and gasping respiration. There was inspiratory collapse of the thoracic cage, and the chest appeared shaped like a roof with the sternum as the ridge pole. Catheter suction, oxygen under pressure, and mouth-to-mouth respiration altered the situation only momentarily, and it was clear that the infant would soon exhaust himself. Chest films revealed a massive bilateral atelectasis, but the tracheobronchial tree contained air out to the finest divisions, being clearly outlined against collapsed pulmonary parenchyma. Bronchoscopy was performed, and the tracheobronchial tree found to be normal; 0.125 per cent neosynephrine was used to wash the bronchi. Following this, there was an immediate change in the shape of the thorax, with improvement in respiration and color. X-rays taken following the procedure showed diffuse partial clearing of the chest.

*Author's instrument.[9]

Fig. 1. Before bronchoscopy.

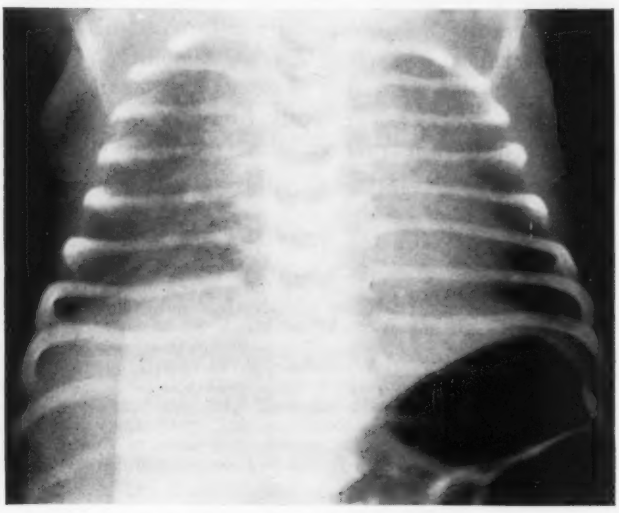

Fig. 1. Immediately after bronchoscopy.

Fig. 1. Complete clearing.

Unfortunately the outcome is not usually so successful as in the case just described, and the atelectatic lung remains unaltered; nevertheless, in the author's opinion the procedure is worthwhile, since in a series of 40 cases, no glottic edema or other complicating factor incident to the instrumentation was encountered, even though a few infants were bronchoscoped several times in a 24-hour period.

H. C. Miller, Edith Potter[10] and others have described a syndrome which, until it is more clearly elucidated, can best be termed "resorption atelectasis with hyaline membrane formation." Labate and others thought that it was formed in the alveoli and alveolar ducts by the residue of nonabsorbable portion of aspirated amniotic sac contents; however, Wilson and Farber have been able to produce experimentally hyaline membranes by hyperventilating the lung and introducing a foreign substance, such as ink or exudate, into the trachea. Bubbles of air become trapped in the alveoli, thus forcing the hyaline substance against the alveolar wall and producing the

typical picture. Farber and Wilson found hyaline membranes in other diseases where extreme respiratory efforts were made because of cyanosis, etc.

Behole, Gibson and Miller[11] found that these lungs could be made to expand by exerting sufficient negative pressure on the lung surface.

Clinically, this disease is thought to be an irreversible process. Thus, James M. Kennedy and Herbert P. Freidman[12] report seven cases of resorption atelectasis with hyaline membrane. Their description is typical:

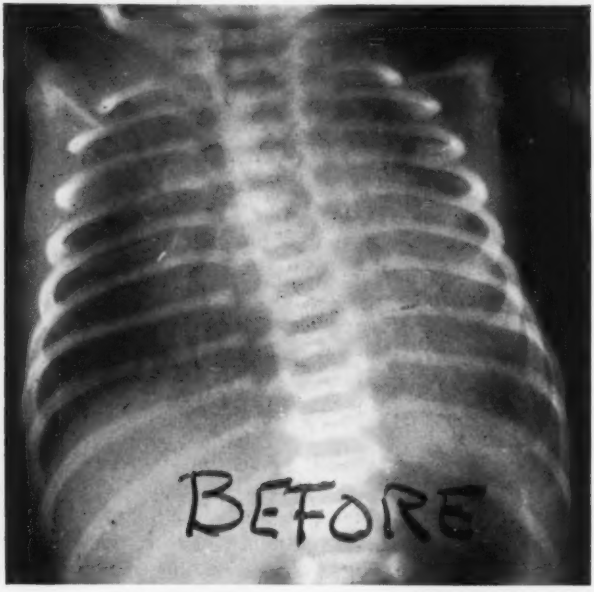

Fig. 2. Before application of traction.

"Briefly, a newborn breathes spontaneously, seems normal to auscultation and percussion, and then within a few hours becomes cyanotic. This is usually relieved by oxygen, but the respiratory rate and effort become more pronounced. The

lower ribs, sternum and abdomen show retraction with each respiration. This becomes more severe, and the cyanosis is no longer relieved by oxygen. Death frequently occurs within the first two days. This entity was formerly considered to result from aspiration of amniotic fluid and vernix caseosa, hence the terms vernix membrane, hyaline membrane and others which have often been appended. The etiology, however, remains obscure."

Recently the author was privileged to treat three cases which clinically resembled this syndrome. Chest films showed a strong resemblance to the chest films taken in Kennedy and Freidman's cases.

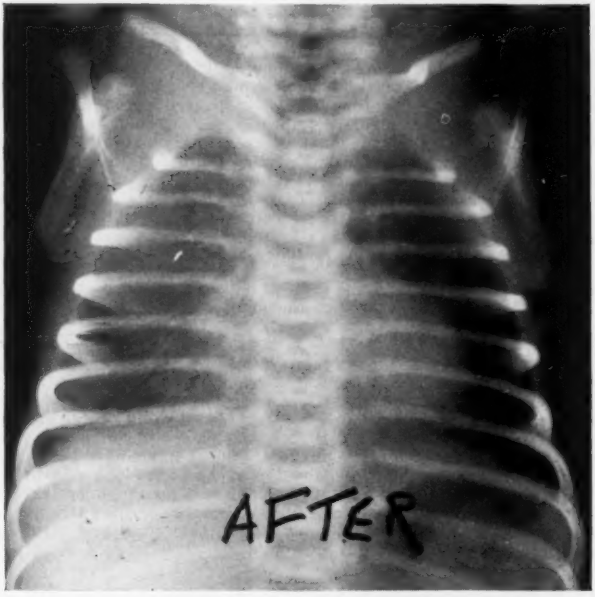

Fig. 2. After traction.

A female infant, weighing four pounds, seven ounces at birth, appeared to be well oxygenated and approximately eight to 10 hours after birth appeared cyanotic. It was placed in an incubator and two days later, when it became apparent that the child would not survive, bronchoscopy

was requested. X-rays before bronchoscopy showed bilateral patchy "fetal atelectasis." It is noteworthy that the tracheobronchial tree was clear on the film. Bronchoscopy confirmed this opinion and the instillation of 0.125 per cent neosynephrine produced no noticeable improvement. Cyanosis was marked and the infant became "gray" and limp, indicating impending exodus. Marked inspiratory retraction of the sternum and lower rib cage persisted. At this point the xyphoid was grasped with an Allis clamp through a skin incision. The clamp was suspended by two rubberbands from the roof of the incubator so that gentle resilient traction was exerted on the lower sternum and rib cage. Improvement was almost immediate and dramatic. The infant appeared well oxygenated in a matter of 10 or 15 minutes. Traction was maintained for 48 hours, and intermittently for 48 hours more. At this time retraction of the sternum had largely disappeared and the child could be removed from oxygen without distress. At the present writing the child has gained weight and appears healthy. Follow-up X-rays showed clearing of the lungs.

The second case was a female infant 30 hours old, weighing three pounds, seven ounces at birth. It was delivered by Cesarean section because the mother was bleeding from a placenta praevia. Upon delivery the child was placed in the airlock because of inspiratory collapse of the chest wall and cyanosis. Bronchoscopy was requested because no improvement was noted after nearly 30 hours. This was performed and no obstructing material located; 0.125 per cent neosynephrine was instilled without improvement. A suture was then passed through the xyphoid and traction exerted. Immediate improvement in color was noted and the baby appeared well oxygenated in room atmosphere. Traction was released, the chest wall again showed the characteristic inspiratory retraction, and cyanosis returned. Traction was then resumed with the color again returning to normal. Traction was maintained for 12 hours, at the end of which time the chest wall showed no retraction and the child appeared well oxygenated. Follow-up chest films were clear, and at this writing the child is gaining weight and is in good health.

The third case, a male infant delivered by Cesarean section, weighed four pounds, 15 ounces, cried spontaneously at birth, and the color was good. Approximately one and one-half hours after delivery the child was placed in the airlock because of cyanosis. It gradually became worse, developing retraction of the lower sternum and rib cage. Finally, after 18 hours, bronchoscopy was requested since the infant's condition was gradually deteriorating. At that time no rales or moisture were heard in the chest. A portable chest X-ray revealed patchy "fetal atelectasis throughout both lung fields." A suture was placed through the xyphoid and strong retraction exerted. There was almost immediate improvement in color. Traction was left in place for 12 hours, at the end of which time the suture was pulled out. The child was so much better that it was not replaced. Retraction was noted only with crying. Cyanosis was gone. Chest film showed partial clearing at this time and in 24 hours almost complete clearing of the lung fields.

While not advocating the universal acceptance of this form of treatment, it is certainly worthy of further trial. It is believed that the explanation of this phenomenon is as follows: The atelectatic lungs were more resistant to the pull of the diaphragm than the soft thoracic cage. The result was

inspiratory collapse or retraction of the chest walls. By exerting traction on the xyphoid, the diaphragm was given something to "fix" against and more of its contractile force was applied to "pull" the lung open. It is possible that the first infant may have had a short central tendon of the diaphragm or funnel chest, although this does not seem likely in view of the rapid improvement which remained even after traction was removed. If this experience proves correct, then one can assume that hyaline membrane formation is the result of mechanical hyperventilation of portions of the already expanded lung in the dyspneic infant, and that "resorption atelectasis" is in reality due to instability of the chest wall, which does not allow enough negative pressure to be exerted at the surface to keep open a very elastic lung.

SUMMARY.

The mechanisms of expansion of the newborn lungs are reviewed. The theories of formation of so-called pulmonary hyaline membrane are discussed.

Four cases of atelectasis in the newborn are presented. One was treated successfully by bronchoscopy alone. The other three, thought to be cases of "absorption atelectasis with hyaline membrane," were treated successfully with sternal traction.

The use of sternal traction is thought to be a new method in the treatment of this condition.

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MYCOTIC INFECTIONS IN OTOLARYNGOLOGY.*

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The study of mycotic infections pathogenic to man is comparatively recent. Although Hooke, as early as 1677, recorded instances of microscopic vegetable forms, it was not until 1910 that Sabouraud's monumental work inaugurated the science of medical mycology to the profession. The interest created by his well written and beautifully illustrated work has continued to the present time. Since Sabouraud's publication, students of mycology have been legion. It is interesting to note that much of the recent work has been done in this country (for example, Benham,[1] Jacobson, Dodge, Henrici,[2] Conant,[3] Littman[4]), and that the majority of them are dermatologists or actually associated with dermatological departments of universities. This is perhaps due to the fact that most mycotic infections have some skin manifestations, but many display lesions that are of interest to the otolaryngologist.

One of the greatest obstacles to the clinical study of mycotic infections is the fact that confusion exists in the medical literature regarding the classification and identification of true fungi. A good working classification for the otolaryngologist is the following:

A. Higher Bacterial Forms (Streptothrix).
  1. Nocardia.
  2. Actinomycetes.

B. Budding Forms (Blastomycetes).
  1. Saccharomycetes.
  2. Torulae.

*Read at the Thirty-eighth Annual Clinical Congress, New York, N. Y., Sept. 22-26, 1952.

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B. Budding Forms (Blastomycetes) (continued).
  3. Oidiae.
  4. Blastomycetes.
  5. Moniliae.
  6. Coccidioides.
  7. Sporothrices.

C. Filamentous Forms.
  1. Aspergilli.
  2. Penicillia.
  3. Mucors.

These fungi, with few exceptions, do not lend themselves to the use of bacteriological techniques for their identification, but they do have some common characteristics as follows: They are all gram-positive; do not ferment sugars; do not reduce nitrates; cannot be characterized by tests such as coagulation of serum, or milk, or by liquefaction of gelatin. They are identified, therefore, by their morphology; macroscopically by the type of colony formation, and microscopically by the type of spores produced. Macroscopically, there are three colony types: yeast, yeast-like, and filamentous. The yeast colony is soft, bacterial-like, and composed of single cell budding forms. The yeast-like colony, although soft and bacterial-like, contains not only single cell budding forms on the surface of the medium but also hyphae which penetrate the medium. The filamentous colony presents the appearance of a typical mold composed of branching hyphae which form into vegetative and aerial mycelium. The spores are produced by the hyphae and are referred to as reproductive mycelium.

HIGHER BACTERIAL FORMS.

The actinomyces and the streptothrix are of medical interest. The latter is a relatively harmless organism which produces warty growths on the lymphoid tissue of the oropharynx or in the tonsillar crypts. Although difficult to eradicate by topical applications alone, streptothrix may be eliminated by cautery, electrocoagulation, or surgical removal of the involved tissues.

Actinomyces possess marked invasive and destructive qualities. Cervicofacial actinomyces account for over 50 per cent

of all cases occurring in man. The lesions spread widely by continuity, producing areas of abscess formation with fistulous tracts. Most often the point of entrance is in the mouth and there are instances of involvement of the ethmoidal and maxillary sinuses for this source (Lewy and Manning[5]). Thoracic actinomyces account for about 15 per cent of the cases. Found principally in the lungs, it produces abscess formation and cavitation; complications can follow, such as emphyema and even eventual perforation through the chest wall. Results obtained by treatment with sulfonamides are somewhat inconsistent. Good results have been obtained with large doses of penicillin; however, some strains of actinomyces differ in sensitivity to it. The use of iodides and thymol as well as X-ray may be tried. Extensive surgical measures may be necessary to remove the infected focus or to provide drainage of abscessed areas.

BUDDING FORMS.

The saccharomycetes or common yeasts do not ordinarily produce disease. In general, it may be said that they are relatively harmless and are of only minor interest to the otolaryngologist.

Torulae (European blastomycosis) produce ulcerative lesions on the mucous membranes. They are prone to invade the central nervous system, causing a highly fatal type of meningoencephalitis. The mucous membrane lesions occur as nodules that break down to form scooped-out ulcers which are quite painful. Diagnosis must be made by histologic methods. The pulmonary involvement produces subacute pneumonitis with low grade fever and mild cough. There is nothing typical in the physical findings or X-ray studies of the chest. Extensive treatment with sulfonamides, when given over a long period, is effective against the mucocutaneous type; good results are also obtained by excision of localized lesions followed by X-ray therapy and iodides by mouth. An especially prepared vaccine, recommended by Benham,[1] may be tried.

Moniliae are oval, budding yeast-like fungi which produce oral infections, particularly in children. They are character-

ized by the typical creamy-white, single, large or multiple small patches scattered over the mucosa. Oral moniliasis responds to alkaline washes or 1 per cent gential violet. The promiscuous use of antibiotics with its disturbance in the biotrophic balance seems to aggravate monilial infections and to complicate their management. This is especially true in the bronchopulmonary type which will respond well to iodides by mouth.

Blastomyces is a spherical, thick-walled budding yeast-like fungus. The disease is usually referred to as North American blastomycosis or Gilchrist disease; however, several fungi have been isolated from the North American types of blastomycosis, resulting in much confusion in the literature. Children have been reported with involvement of the tongue with blastomyces. Erich[6] reports a case involving the oral cavity and nose, cured by electrocoagulation, iodides, and X-ray. The lungs are the most frequent site of infection, but the laryngeal type is not uncommon, as has been reported by Jackson.[7] Sulfonamides and penicillin seem to have no effect on the course of the disease. Mucous lesions are best treated with iodides by mouth and X-ray directed at the spreading border of the lesion.

Coccidiodes immitis is a spherical, thick-walled endospore-filled organism found in exudates and a fluffy cotton-like fungus when grown at room temperature. It is probably the most dangerous of the budding forms. Superficial lesions occur chiefly at the nasolabial folds and while essentially chronic in nature, are not dangerous to life as long as they remain localized. When systemic involvement occurs, the lungs as well as other viscera and the central nervous system become involved, and prognosis is poor. Symptomatic treatment with bed rest, high vitamin, high caloric diet may be useful. There is no specific treatment.

FILAMENTOUS FORMS.

The aspergilli, penicillia, and the Mucors produce lesions somewhat similar in appearance. The aspergillus is characterized by its appearance in many parts of the body, but the

ear is the most common site of infection. This type of mycotic infection is stressed here because it is most frequently encountered by the otolaryngologist. It is a minor aural disease and is not, as a rule, of a serious nature. The fungi produce intense itching in the course of their growth, and cause epithelial exfoliation which fills the external auditory canal. Sometimes the white filamentous growth is visible and, if sporulation has occurred, the black color of myriad spore capsules gives the appearance of coal dust in the canal. Diagnosis can be substantiated by cultures. Biopsy may be necessary in questionable lesions, but this should be cautiously undertaken.

ROUTINE OFFICE DIAGNOSIS.

We have adopted a routine method for study of mycosis of the external ear which, although lacking the precision of more elaborate procedures (Gill[8]), is a practical one that is adapted to office practice. Under aseptic conditions a sterile swab is carefully introduced into the external canal and plated on Littman's oxgall agar or Sabouraud's media; the former is preferred. Then, from the same swab microscopic slides are made and stained with gram and giemsa stains. These are studied microscopically for bacteria and for cell identification, principally eosinophile. A strip of nitrazine or alkacid paper is carefully inserted into the canal for a few minutes, then removed and compared with a pH color chart. This will in a practical way establish the acidity or alkalinity of the secretion and will help as a guide in the selection of the proper drug to be used. The majority of cases will be within the alkaline range. These results seem to indicate that in mycotic ears the drainage is definitely alkaline and the normal acid cloak of the canal is altered.

TREATMENT.

Once the diagnosis is established, treatment should be instituted with the drug of choice. There are many currently popular drugs; the relative merits of many are questionable. One might mention a long list of such medicaments, the claims of their effectiveness in all infections of the ear have little scientific basis. We have investigated a number of prep-

arations recently placed on the market to determine their efficacy in the treatment of fungus infections in the external ear. Among these may be mentioned: sulfamylon, undecylinic acid, zinc caprylate, sodium caprylate, sulphur dioxide solution, isoparaffinic acid, furacin, dalyde, polymyxin B sulphate, and others. A new solution, containing aerosporin, thymol, acetic acid and a similar one with cresatin seems to offer definite possibilities in the treatment of external otitis complicated by fungi.

No one treatment suffices for all mycotic infections. Iodine or the iodides apparently possess the greatest degree of effectiveness when applied locally or taken internally. Thymol also ranks high in effectiveness. We are still partial to cresatin in the initial stage of the infection as it contains many desirable qualities, being analgesic, fungicidal, and bacteriocidal. Radiant energy such as ultraviolet light, X-ray, and radium have all been tried, and of these X-ray seems to be the most valuable. Filtrates made of a mixture of trichophytin, oidiomycin and epidermaphytin can be utilized in certain cases of dermatomycosis of the ear canal and auricle as well as in cases of monilial stomatitis with satisfactory results. Low dosage intracutaneous methods of desensitization is, in our opinion, of definite value.

SUMMARY.

Modern diagnosis and treatment of mycotic infections as they relate to the otolaryngologist is presented.

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ENDOTRACHEAL ANESTHESIA IN CHILDREN.*†

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There still exists considerable controversy in regard to the use of endotrachael anesthesia in children. At a recent visit to medical centers in the East, I was surprised to find a certain reluctance for this form of anesthesia; this observation, and especially an article by Shirley Baron,[1] of San Francisco, prompted me to make this presentation. In 1950, he reported the results of a study of 80 consecutive patients who had endotracheal anesthesia. Among these were eight cases of laryngeal obstructions with two deaths and seven cases of laryngeal granuloma; that is, 18 per cent of the cases showed laryngeal sequelae following endotracheal anesthesia. He stated, "It is difficult for us to understand why, in children, endotracheal anesthesia should be used for an adenotonsillectomy in any instance." The progress made in the last decade in the field of anesthesiology has benefited surgery immensely as such, but particularly head and neck surgery. Many surgical procedures which are routine performances today would have been adventurous undertakings a decade ago when our present type of anesthesia was not available.

Adenotonsillectomy is the most frequent operation performed. The U. S. Health Department estimated that one and one-quarter million tonsillectomies are performed every year in the U. S. A., and about one million of them are done under general anesthesia. The administration of anesthesia for tonsillectomy is one of the most difficult tasks for the anesthetist, and 75 per cent of all fatal accidents in tonsillectomy are due to faulty anesthesia. Asphyxia due to obstruction of respira-

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tion is the most common and serious complication of anesthesia. No other operation is more apt to cause obstruction to respiration than tonsillectomy. We work next to the respiratory tract in a small cavity, with the head hyperextended. Faulty use of the tongue depressor with blood and excessive mucus in the oral cavity may cause serious respiratory embarrassment. It is a constant challenge for the anesthetist and operator to maintain adequate anesthesia and at the same time an open airway. The threshold between sufficient relaxation and respiratory depression in children is small; suboxygenation over a period of time may lead to permanent cerebral damage. The maintenance of an open airway is paramount to any form of safe general anesthesia. Safe anesthesia, especially in tonsillectomy, can be provided only if an endotracheal tube is utilized. Safe anesthesia means the ability to maintain adequate oxygenation at all times.

We have used this form of anesthesia in all our tonsillectomies in children for many years to our greatest satisfaction, without encountering any serious complications. In teaching institutions where internes and residents perform a great many of these operations, the time element must be considered; to maintain an even plane of anesthesia over a longer period of time is still more difficult without the benefit of an endotracheal tube. I am convinced many fatal accidents have been prevented in these instances by intubating the child patient.

Dr. Knight, Director of the Department of Anesthesiology at the University of Minnesota, and his associates are ardent advocates of endotracheal anesthesia and have contributed a great deal to its perfection. Under the guidance of his associates who head the Department of Anesthesiology at the Miller Hospital in St. Paul, all adenotonsillectomies since 1949 were performed under endotrachael anesthesia. We reviewed 410 cases of adenotonsillectomies performed from 1949 to 1952 in which the endotracheal technique was employed. The anesthesia was always under the supervision of Dr. Cohen or his associate, Dr. Paulson. In 75 per cent of the cases the intubation was done by one anesthesiologist. The

fact that these cases were under the same supervision and a well established technique was routinely applied is undoubtedly a contributing factor in achieving such satisfactory results.

Certain rules must be followed in order to obtain a successful endotracheal anesthesia:

1. Proper depth of anesthesia and relaxation of jaw muscles are essential for passing an endotracheal tube without causing trauma to the patient.
2. Selecting the proper size and length of the tube is of greatest importance.

If the tube is too long it may enter one of the main bronchi and so prevent proper ventilation from the opposite lung and may lead to lung collapse. An oversized tube is apt to traumatize the larynx, which might lead to laryngeal edema, contact ulcer, or granuloma. The following table suggests the sizes of tubes used for the different age groups:

TABLE 1. ENDOTRACHEAL TUBE SIZES.

Size No.	Age
2	2 years
3	3- 4
4	5- 7
5	8-10
6	11-15, small adults
7	16 and older

During 1949, Magill rubber tubes were used, and in 1950 these were replaced by polyvinyl "Portex" plastic tubes. Any laryngoscope, with which adequate exposure of the larynx can be obtained, is suitable for intubation. The medium-sized "Wis-Foregger" blade with hook-on handle has been used in our group and is suitable for almost any age.

Various anesthetic agents have been used in our group for the induction and maintenance of anesthesia. In our early series we utilized ether with vinethene, cyclopropane with pentothal, or cyclopropane ether, while during the last two years, pentothal-curare with nitrous oxide was used in most

cases. If a competent anesthesiologist is on hand, I feel that the choice of which anesthetic agent should be used in each particular case should be his. On the other hand, if only a nurse anesthetist is available and the otolaryngologist does the intubation himself, ether-oxygen combination is still the safest anesthetic agent. Whichever anesthetic is used, it is of greatest importance that the pharyngeal and laryngeal reflexes are obtunded completely, when the intubation takes place. Only in this plane of anesthesia is an atraumatic intubation possible. Blind intranasal intubation should be discouraged except in particular cases, where oral intubation is of disadvantage. In most of our cases, orotracheal intubation was carried out.

In the beginning the operator may object to the endotracheal tube occupying so much space in the oral cavity, especially in the presence of abnormally large tonsils, but one can adapt to the tube very quickly if it is shifted from one side of the mouth to the other to suit the needs of the surgeon. Intranasal intubation interferes somewhat with the performance of adenoidectomy, but if necessary it can be done satisfactorily by shifting the tube sideways and forward.

Two groups of cases were studied in which endotracheal anesthesia was applied; one group comprises 410 cases of adenotonsillectomies performed at the Miller Hospital in the years 1949 to 1952; the second group includes 153 cases of adenotonsillectomies performed at the University of Minnesota hospital in the same years:

GROUP I.

CHARLES T. MILLER HOSPITAL: 410 ADENOTONSILLECTOMIES.

TABLE 2. AGE GROUPINGS OF PATIENTS STUDIED.

	Number of Cases	% of Cases
1- 5	144	35
6-10	188	46
11-15	48	12
16-20	14	3
20 years plus	16	4

TABLE 3. ANESTHETIC AGENT EMPLOYED.

	Number of Cases	% of Cases
Ether (Vinethene induction—64)	66	16
Cyclopropane (Pentothal induction—4)	43	10
Cyclopropane-Ether	24	6
Pentothal-Curare N_2O	277	68

TABLE 4. DURATION OF ANESTHESIA.

	Number of Cases	% of Cases
0-15 Minutes	6	2
16-30 Minutes	115	28
31-45 Minutes	164	40
46-60 Minutes	96	23
61 minutes plus	29	7

TABLE 5. ENDOTRACHEAL TUBES USED.

	Number of Cases	% of Cases
Oral—Tracheal	362	88
Nasal—Tracheal	48	12

TABLE 6. DURATION OF HOSPITALIZATION.

	Number of Cases	% of Cases
1 Day	16	4
2 Days	362	88
3 Days	15	4
4 Days plus	17	4

TABLE 7.
POSTANESTHETIC COMPLICATIONS NOTED DURING HOSPITAL STAY.

	Number of Cases
Pneumonia	0
Atelectasis	0
Hoarseness	1
Tracheotomy	0
Laceration of pharynx	0
Broken or missing teeth	0

The University Hospital is primarily a teaching institution. The anesthesia in most cases is given by residents of the Department of Anesthesiology and some complications might be expected in this group, due to the lesser experience of the attending anesthesiologist as well as the surgeon, who in most of these cases is a resident of the Department of Otolaryngology.

GROUP II.
UNIVERSITY OF MINNESOTA HOSPITALS:
153 ADENOTONSILLECTOMIES.

TABLE 8. AGE GROUPINGS OF PATIENTS STUDIED.

	Number of Cases	% of Cases
1- 5	58	38
6-10	65	42
11-15	26	17
16-20	4	3
20 Years plus	0	0

TABLE 9. ANESTHETIC AGENT EMPLOYED.

	Number of Cases	% of Cases
Vinethene-Ether	20	13
Ether	6	4
Cyclopropane	0	0
Pentothal-Cyclopropane	5	3
Pentothal-Curare	122	80

TABLE 10. DURATION OF ANESTHESIA.

	Number of Cases	% of Cases
0-15 Minutes	1	1
16-30 Minutes	9	6
31-45 Minutes	46	30
46-60 Minutes	48	31
61 Minutes plus	49	32

TABLE 11. ENDOTRACHEAL TUBES USED.

	Number of Cases	% of Cases
Oral—Tracheal	148	97
Nasal—Tracheal	5	3

TABLE 12. DURATION OF HOSPITALIZATION.

	Number of Cases	% of Cases
1 Day	117	76
2 Days	14	10
3 Days	11	7
4 Days plus	11	7

TABLE 13.
POSTANESTHETIC COMPLICATIONS NOTED DURING HOSPITAL STAY.

	Number of Cases
Pneumonia	0
Atelectasis	0
Hoarseness	1
Tracheotomy	0
Laceration of pharynx	0
Broken or missing teeth	0

Altogether, 563 cases were studied, and the only complications noted were two cases of hoarseness; certainly not a serious after-effect. If any complications such as cord granuloma or ulcer had developed after the discharge of the patient from the hospital, in 99 per cent of the cases it would have come to the attention of the attending physician. None were reported. These statistics sound so good that one might doubt their veracity. It certainly proves endotracheal anesthesia is a safe procedure, even in small chidren, if applied by an anesthesiologist or otolaryngologist who has been properly trained in its administration. There is no other form of anesthesia at present which can replace it and not sacrifice the all important factor in general anesthesia, that is, to maintain an open airway during its entire course. The only really serious complication which may occur, laryngeal obstruction as a sequelae of endotracheal anesthesia, can, in 99 per cent of the cases be avoided, if the administrator of anesthesia follows the essential prerequisites for this type of anesthesia. In my opinion, there is no question but that this form of anesthesia has prevented many serious or even fatal complications in our field of surgery; therefore, it seems mandatory that every otolaryngologist should familiarize himself with this type of anesthesia and make use of it on every suitable occasion. We all should strive for greater perfection of this method of anesthesia and not abandon it on account of an occasional sequela which in most cases is not serious.

I want to close this presentation, quoting the father of endotracheal anesthesia, who has done so much to perfect and popularize it, namely, Dr. Paluel J. Flagg,[2] who in the discussion of Baron's paper stated: "My impression is that there is no substitute for this technique but that the details of its common usage may well be improved. In other words, instead of eliminating the technique, improve its use. Let us consider the following: The shadow of impending death from aspiration and from asphyxia falls over all surgery of the head and neck performed under general anesthesia. This shadow — this hazard — is eliminated by the endotracheal tube. In the present state of our knowledge, the use of the

endotracheal tube for this type of surgery is mandatory. There is no other mechanical means of protection available. Indeed, a failure to use this protection in the case of accidental death from asphyxia or from aspiration may shortly be regarded as malpractice."

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MEDICAL SERVICES TO THE SPEECH AND HEARING HANDICAPPED IN TENNESSEE.*†

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In order that you may share our philosophy relating to the impact that faulty communication exerts upon the life adjustment of individuals so handicapped, I describe briefly two patients:

A. B., a nine-year-old white male, was born with a cleft palate which was satisfactorily closed surgically before the age of two. The child's speech pattern, however, was faulty. He entered public school at six, repeated the first grade once, and then after being promoted to the second grade was forced to repeat it also. It was early in this repetition of the second grade that he came to our attention. Careful investigation revealed a bright boy whose teachers felt compelled to fail him because his speech was unintelligible and they, therefore, could not ascertain his intellectual ability. Speech training was begun at once and at the conclusion of the school year, the child had made such improvement that the school authorities were considering promotion from the second to the fourth grade. Without speech training this child was destined most assuredly to a drab and menial existence.

The second patient, L. A., age 12, was also a seemingly alert and intelligent boy of good parentage and environment, whose school and community adjustment was marked by such phrases as "a severe emotional problem," "a behavior problem," "incorrigible" and "inattentive." His educational adjustment had been exceedingly stormy. When a team of audiologists from the speech and hearing program visited the school where this patient was in attendance, as if by Providence, his classroom was one of those being tested, and this boy was found to be severely hard of hearing. Immediate medical examination was made by one of our otolaryngologists, following which a hearing aid was fitted at the clinic. Needless to say this boy has made an exemplary record since receiving and being trained to use his hearing aid, and a life that seemed certain for continuous antisocial acts has been transformed into normal and useful endeavors.

Such problems as these had weighed heavy upon the mind of Dr. W. W. Wilkerson. He had long appreciated the seri-

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†From the Division of Maternal and Child Health Services, Tennessee Department of Public Health.

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ousness, the magnitude and the universality of the problem and had observed that the uncoordinated efforts of physicians, laymen and communities were ineffectual. Prompted by conscience and an inexhaustible public spirit, he began in 1949 to interest members of the medical profession, leaders in the field of public health and private citizens and organizations, in the problems of those with speech and hearing defects, and in 1950 the Tennessee Hearing and Speech Foundation was chartered under the laws of Tennessee, wholeheartedly supported by the Tennessee State Medical Association, the Academy of Ophthalmology and Otolaryngology and the Nashville Academy of Medicine, which group annually appoints a medical advisory committee to guide the local Hearing and Speech Center in its medical affairs.

Immediately after its inception, the Foundation began to study the overall needs of Tennessee in the field of hearing and speech, divided the program into three essential component elements and therafter set about the details necessary to realize each. Early, a clear line of responsibility was drawn between medical services and educational services, such that case finding, diagnostic screening, medical service and the services obtainable in the hearing and speech clinics were declared medical and the education of children purely educational.

The first component element, or Phase One, case finding and diagnostic screening, was designated the responsibility of the Tennessee Department of Public Health, Crippled Children's Service, one of the actively cooperating organizations of the Foundation. To make this possible, the Tennessee Hearing and Speech Foundation, with appropriate medical support, brought the matter to the attention of the Tennessee General Assembly in 1951, which body passed legislation which recognized the speech or hearing handicapped child as a crippled child, and appropriated $120,000.00 annually to the Tennessee Department of Public Health for this work. The latter agency's Crippled Children's Service is accomplishing the case finding by means of highly qualified teams of audiologists and speech pathologists testing children in all parts

of the state.[1,7] Medically indigent children become wards of Crippled Children's Service for subsequent necessary services.

A basic and important premise of the Foundation is that every person must have an examination by an otolaryngologist, together with whatever medical treatment is indicated, prior to his being accepted by a hearing and speech clinic;[2] therefore, the screening teams or physicians who have established the existence of a defect, refer the patient to an otolaryngologist for ear, nose and throat study. The otolaryngologist, upon suspicion that a given patient needs general medical care, plastic surgery, pediatric, orthodontic, psychiatric or other medical or dental care, refers that patient for indicated treatment. This mechanism serves to extend the knowledge of and the interest in the program to all related professional groups and has in fact greatly broadened and strengthened support from the profession as a whole. We feel that many of the hearing and speech disorders of young children can be corrected by good medical care. When all medical treatment has been completed the patient is then referred to the hearing and speech clinic or center if his disorder of speech or hearing persists. Children who have benefited by such treatment but have a minor residual speech defect are referred by the otolaryngologist to the local superintendent of schools, whose speech teacher will be available to complete the rehabilitation and coordinate the educational needs of the child. Patients under care of the hearing and speech centers are referred back to private physicians and referring otolaryngologists periodically for follow-up and also when and if any indication for medical treatment arises.[3] All of the latter above mentioned services constitute the second component element or Phase Two of the program. There are more than 45 otolaryngologists in 16 cities and towns cooperating with the Tennessee Department of Public Health and the Tennessee Hearing and Speech Foundation at this time (see Fig. 1).

The development of specialized hearing and speech clinics or centers was also a necessary responsibility of the Foundation. The Bill Wilkerson Hearing and Speech Center in Nashville was the first of these. This center, working cooperatively

with the Tennessee Department of Public Health, the Nashville-Davidson County Health Department, Vanderbilt University, Peabody College for Teachers and the Nashville community, provides all the special services necessary in the rehabilitation of the speech and hearing handicapped. It also conducts a training program in audiology and speech pathology leading to a Master's degree awarded by Vanderbilt University School of Medicine, and conducts a classroom for pre-

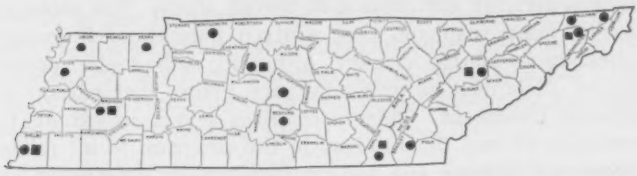

Fig. 1. Geographic distribution of cooperating otolaryngologists in relation to hearing and speech centers.
● Cooperating otolaryngologists. ■ Hearing and speech centers.

school and school age hard-of-hearing children in cooperation with the local city and county public school administrations. In addition to the Bill Wilkerson Center in Nashville, there are five other centers in operation or in process of establishment, organized as chapters of the Tennessee Hearing and Speech Foundation and so located geographically that they are within reach of every citizen of the state (see Fig. 1). It is significant to point out that before any of these chapters were organized, the purposes and plans of the Tennessee Hearing and Speech Foundation were presented to the respective local medical society, so that in every area the local medical society became a sponsoring organization for the local speech and hearing center.

The third component element or Phase Three is concerned with education and is the designated and accepted responsibility of the State Department of Education, which provides local speech and hearing teachers, establishes special instructional programs and provides special classes where indicated.[3] The Tennessee State Department of Education for several years has been slowly building a public school program in

speech and hearing therapy. The general support and the medical supervision necessarily inherent in this type of program was consistently lacking so that the efforts of the Department of Education were rendered less effective than was desired. The Tennessee Hearing and Speech Foundation and the Tennessee Department of Public Health are working with the educators to strengthen and broaden these essential services.

The intimate coordination of this three-phase endeavor has made an extensive service and training program possible.[4,7]

I should like to define the criteria used to establish the existence of a defect before presenting brief statistical data which demonstrate the magnitude of the problem of the speech and hearing defective in Tennessee.[5] A noticeable speech defect is considered minor if it is without observable implications of maladjustment and considered major if it calls attention to itself and interferes with adequate communication or may be causing maladjustments of the individual. In determining hearing defects, all children are screened individually at 15 db in frequencies 250 to 8,000 cycles per second. Those failing this screening are retested to determine the extent of the defect and are classified then as minor if this loss does not interfere with perception of communication and if there is no evidence of potentially progressive disease, and major if hearing is impaired below 15 db in any frequency between 250 and 8,000 cycles per second and/or if there is a history or evidence of chronic pathology.[7]

An appreciation of the magnitude of the problem of the speech and hearing handicapped person in our population may be gained through studying case finding surveys in 19 counties of Tennessee among elementary school children. The percentages of children with speech and hearing defects (major and minor) are shown in Fig. 2. The percentages with respect to hearing do not differ greatly from reported findings of a comparable survey in Harford County, Md.[6]

The first method, a mass survey made in six counties by Public Health Department speech and hearing consultants,

testing approximately 15,000 children, revealed 8.9 per cent of children with speech defects and 13.8 per cent with hearing defects. In all, 21.04 per cent had either speech or hearing

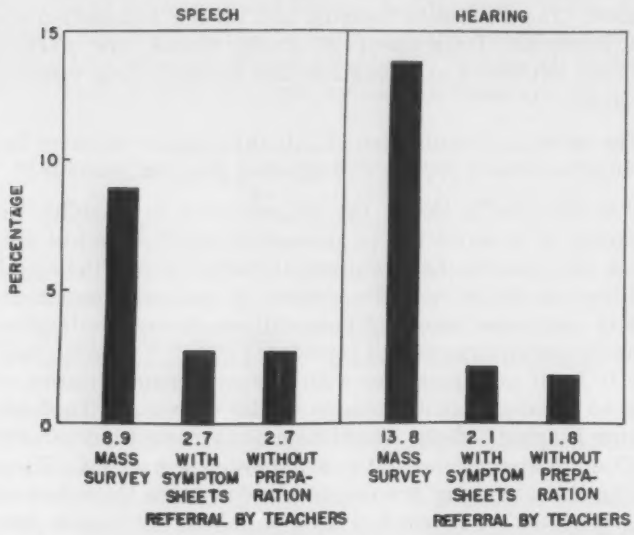

Fig. 2. Percentage of elementary school children with speech and hearing defects according to method of case-finding in 19 counties of Tennessee, 1951-1952.

defect requiring observation, special education, or medical or clinical treatment.[7] Other case-finding methods using teacher referral revealed much smaller percentages of children with defects.

Considering only major defects, using the criteria outlined above, by mass survey there were 1.5 per cent of children found to have major speech defects and 3.3 per cent of children found to have major hearing defects. Case-finding methods using teacher referral again revealed appreciably lower percentages of children with speech and hearing defects as compared with mass survey by trained audiologists and speech pathologists (see Fig. 3).[7]

It seems valid to assume that only through complete mass survey by competent trained workers will the true problem be revealed, and that by less effective methods of case finding, many children with speech and hearing defects will not be recognized early.[7]

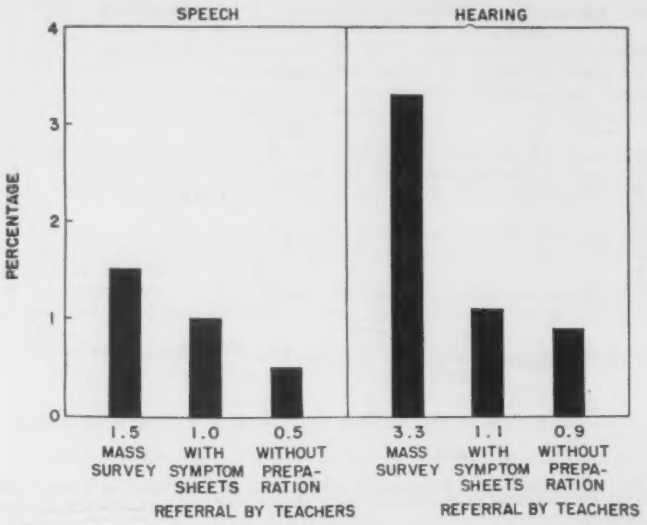

Fig. 3. Percentage of elementary school children with major speech and hearing defects according to method of case-finding in 19 counties of Tennessee, 1951-1952.

Fig 4[7] shows the relation of speech and hearing defects to age, based upon mass survey findings. It is important to point out that total speech defects, high in the early school age groups, tend to diminish slowly as age increases. Early treatment of these would certainly hasten the fall of this diminishing curve so that speech defects would not carry over into the upper elementary grades. It would be expected that this treatment would also improve the school adjustment and the academic progress of these children. Major speech disorders, however, remain fairly constant, indicating certainly the major speech defect needs aggressive attention. Contrastingly,

total hearing defects tend to increase with age as do also major hearing defects. This would seem to support the contention that it is important to find hearing defects early and treat them well, and further, that lack of care in early age compounds the problem as time progresses.[7]

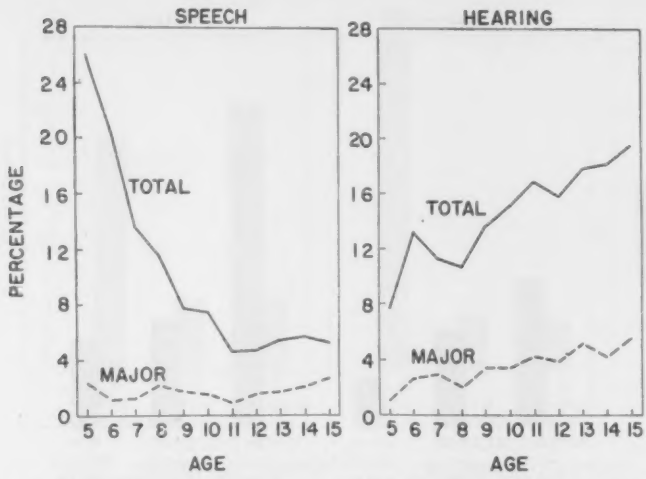

Fig. 4. Percentage of elementary school children examined in a mass survey in six counties of Tennessee with speech and hearing defects, by age, 1951-1952.

In conclusion, there has been presented the development of a statewide program in speech and hearing, growing out of the establishment of the Tennessee Hearing and Speech Foundation. The program is designed to find, treat and rehabilitate persons, especially children, suffering from disorders of communication. It has been emphasized that this entire development has grown out of the recognition on the part of the medical profession that unassisted efforts of individuals and uncoordinated efforts of communities were not effective in meeting needs that were known to exist and has since been demonstrated. Further, it has been emphasized that Tennessee's program has been developed, sponsored and fostered by practitioners of medicine, wherein lies its completeness, its

quality and its strength. It is this kind of leadership and spirit of teamwork with laymen and other organizations that has brought achievement and dignity to the hearing and speech program of Tennessee.

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SPECIFIC PROBLEMS IN THE USE OF ANTIBIOTICS IN OTOLARYNGOLOGY.*

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The changing pattern of some bacteria in their susceptibility to various antibiotics, the nature of specific toxicities and allergic manifestations during and following antibiotic therapy, and the existence of antagonism and synergy between the various antibiotics, are problems common to all fields of medicine. Certain problems, however, primarily concern the otolaryngologist, and these are often difficult and vexing. Entirely new criteria for diagnosis and treatment must be evolved when the otolaryngologist first sees a patient who has received antibiotic therapy for days, weeks or even months.

With increasing frequency, we are faced with *the problem of hearing loss resulting from the indiscriminate use of antibiotic therapy;*[8] in fact, the problem of preventing hearing loss following acute otitis media has become more complex since the introduction of antibiotic agents. The discovery of residual hearing loss of 30 to 50 db throughout the speech range following apparent recovery from acute otitis media appears to be a paradox when we consider that with early, adequate and properly directed antibiotic therapy, damage to the acoustic mechanism is minimized, and spontaneous perforations of the tympanic membrane are either rare or close readily when pyogenic organisms alone are involved. The present rarity of surgery for acute mastoid infections is ample evidence that proper antibiotic therapy favorably alters the course of middle ear infections and, therefore, should conserve the physiologic function of the acoustic mechanism.

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The erroneous and pernicious idea that antibiotic therapy has rendered paracentesis obsolete under all circumstances is prevalent among many pediatricians and practitioners. Such indiscriminate use of the antibiotics inevitably leads to a high incidence of permanent acoustic loss. While paracentesis is rarely necessary with early, adequate and properly directed antibiotic therapy, inadequate, improperly directed or late therapy permits the collection of pus under pressure within the middle ear and the disorganization of the structures of the tympanic membrane and middle ear. Acute otitis media is often a surgical emergency, and as in all surgical emergencies, the indiscriminate use of the broad-spectrum antibiotics is dangerous without careful observation of the patient and exact diagnosis. The distortion of symptoms and objective findings, the reduction of fever, and often the presence of a normal leucocyte count, foster a false sense of security, while lysis of soft tissues and bone, and thrombosis of vessels progress. In otitic infections, masking of symptoms not only leads to hearing loss but also delays the recognition of such other surgical emergencies as subperiosteal abscess and brain abscess.

The ear mechanism does not necessarily return to normal merely because the fever is reduced and pain disappears. In suppurative otitis media, drainage of pus under pressure continues to be a sound therapeutic procedure, if for no other reason than to protect the conduction apparatus. When, in spite of antibiotic therapy, frank bulging of the eardrum persists for more than 24 hours, paracentesis is imperative if serious damage to the conduction apparatus is to be avoided. The dangers of delayed drainage can be demonstrated by audiometric studies, and this evidence is unequivocal and convincing.

When drainage through the eardrum is inadequate, the incision or the perforation must be enlarged. Unless these precautions are taken, the otologist is brought into the picture after pain and fever have persisted for three or four weeks, during which time the discharge has continued unabated. The

suppurative focus and the diseased tissues about it are no longer amenable to antibiotic therapy, and only mastoidectomy will prevent complications or preserve life.

Progressive disease or acoustic loss, or both, can persist after the apparent cure of otitis media by antibiotic therapy. Unconquered infections in the middle ear or mastoid cells announce their presence not long after the discontinuance of therapy, but acoustic loss, particularly in children, may go unrecognized for a very long time. In younger children, hearing defects are especially serious, and effective remedial measures depend upon the earliest possible recognition of not only the acoustic loss but its causes as well.[8] Faulty immunologic responses or abnormal pharyngeal lymph tissue fail to disappear with antibiotic therapy.

Every case of otitis media is a potential deafened patient. The clinician is responsible not only for the restoration of an acutely ill child or adult to health but also for the preservation of function of the hearing mechanism.[8] The limitations of antibiotic therapy in the treatment of otitis media must be emphasized by the otologist. In addition, it should be made a rigid rule that a careful examination of the ears, including audiometric examination, be made soon after apparent recovery from otitis media. This will prevent much disability if the early recognition of hearing loss is followed by prompt and appropriate therapy.

The *appearance of new and unusual infections in the field of otolaryngology is another result of antibiotic therapy.* There has been an increasing incidence of moniliasis, and of proteus and pseudomonas infections in association with the administration of the wide-spectrum antibiotics.[6] Even such rare causes of human disease as the species aspergillus and gram-negative bacteria, such as E. coli, have appeared as the predominating organisms in the nasal passages, paranasal sinuses, oral cavity, pharynx or larynx, and their extension to the lower respiratory tract presents an ever present danger.[15]

With increasing frequency, penicillin-resistant staphylococci are being isolated in chronic paranasal sinus infections.

The newer wide-spectrum antibiotics are more prone to produce oral lesions and other side reactions than is penicillin.[10] Antibiotic reactions most frequently involve the mouth, throat and glossitis, and black, hairy tongue are not uncommon. More and more cases of true thrush are being found in the aged and debilitated.

The development of resistant or nonsusceptible flora must be anticipated as a regular consequence of antibiotic therapy. Since areas normally inhabited by microorganisms are never sterilized by such therapy, it is not surprising that the resistant organisms eventually establish a new or unusual flora, which in turn may produce a new disease. An entirely new flora may result from the suppression of competitive organisms, the implantation of organisms from outside contactant sources, or the acquisition of resistance by biologic adaptation and variation on the part of originally susceptible organisms.[5]

Unfortunately, it is not always a simple matter to determine whether lesions coinciding with the appearance of a new flora are due to infection or whether they merely represent reactions to antibiotic agents. The lesions are sometimes an expression of antibiotic-induced vitamin B deficiency, sensitivity-induced tissue changes as in allergy, or simply a primary irritation following the use of oral antibiotic lozenges. In fact, more alteration in the oral flora occurs when antibiotic troches are dissolved in the mouth than when the same drugs are administered by other routes.[5]

The appearance of C. albicans (monilia) in the upper respiratory tract, following antibiotic therapy, has received wide attention.[6] Here we are dealing with an endogenous organism, frequently found to be a normal constituent of the respiratory and gastrointestinal flora,[1] of which it represents but a small part. In persons receiving wide-spectrum antibiotics, C. albicans can be recovered in large numbers and often in the total absence of symptoms. Antibiotics do not directly stimulate the growth of fungi but fungi are intrinsically resistant to all the known antibiotic agents. They flourish when antibiotic agents convert a predominantly gram-

positive, susceptible bacterial flora to a predominantly gram-negative resistant flora. The mere presence of fungi does not warrant the conclusion that there has been actual invasion of tissues.

The definitive diagnosis of moniliasis requires more than the demonstration of C. Albicans, with budding cells and filaments, in smears from the oral cavity;[5] however, when their presence is associated with definite lesions in the form of whitish films or plaques, resembling coagulated milk, readily detached from inflamed, bleeding mucous membrane, we are dealing with the typical lesion of thrush.

Problems arising from the administration of the wide-spectrum antibiotics include *1.* the recognition of the presence of an entirely new or bizarre microorganism or flora; *2.* the interpretation of a new and unfamiliar clinical picture; *3.* the relationship of a new flora to demonstrable pathologic lesions, and *4.* the development of effective therapeutic methods for the control of these new infections in our field.

Structural damage to body tissues occurs as a result of antibiotic therapy. The otolaryngologist is particularly concerned when damage involves the special senses of smell and taste, or impairs the hearing and labyrinthine functions. It has long been recognized that severe damage to the vestibular and cochlear mechanisms may arise from the therapeutic use of streptomycin and dihydrostreptomycin. A report of the Council on Pharmacy and Chemistry of the American Medical Association has pointed out that of 543 tuberculous patients, who were given 2 gm. or more of streptomycin daily, 1.1 per cent developed deafness and 96 per cent developed vestibular symptoms; however, it is now evident that deafness from streptomycin is unlikely if the total daily doses are less than 3 gm.

The incidence of cochlear and vestibular complications is directly proportional to the size of the daily dose of streptomycin or dihydrostreptomycin and to the number of days such daily doses are administered.[11] These complications are always bilateral and appear more abruptly with dihydro-

streptomycin and more gradually with streptomycin.[12] Improvement may follow slight involvement of the cochlear and vestibular apparatus, but severe damage is permanent and irreversible. When deafness occurs, it is of the perceptive type, and preceded by tinnitus. Dihydrostreptomycin impairs the cochlear mechanism more readily than streptomycin but is less injurious to the vestibular apparatus. Thus, 30 to 40 per cent of individuals receiving dihydrostreptomycin acquire perceptive deafness of 30 or more 4b, and the symptoms have appeared after therapy had been discontinued for as long as 24 days.

The site of damage to the vestibular and cochlear mechanisms, when these two drugs are used therapeutically, is in dispute. Various investigators have demonstrated in both human and animal material that damage occurs in the end-organ, while others have shown that damage occurs in the central portions of the vestibular and cochlear mechanisms.[4,7,14] At the present time we must assume that these two drugs are capable of producing destructive tissue changes in both the central and the peripheral portions of the vestibular and cochlear mechanisms.

During the first three months of gestation, the auditory apparatus of the fetus is particularly vulnerable to damage. The rôle played in such damage by the transplacental infections—spirochetal, viral and influenzal—and by quinine and other drug intoxications is fairly clear. Are antibiotics a potential cause of prenatal acquired deafness?

The spectacular multiplication of available antibiotics, and the increasing use of combinations of these drugs, prevent a satisfactory answer to this question. It has been established that streptomycin crosses the placenta, and, therefore, when administered to pregnant women, it reaches the rapidly developing acoustic mechanism of the fetus. Although mothers with active tuberculosis and treated with 1 gm. of streptomycin daily for three or four weeks preceding delivery, have been delivered of infants with normal vestibular and cochlear function, the effects of similar dosage during the first three months of pregnancy are still unknown.

The ability of certain antibiotics to produce degenerative changes in the central nervous system or in the sensory end-organs may be capable of therapeutic application. Thus, streptomycin, because of its tendency to damage the vestibular apparatus, has been used in the treatment of Ménière's disease because it seemed to offer the same results as surgery without its risks.[2] The work of Winston and others, however, has demonstrated the potential dangers of the use of streptomycin for this purpose.

Past experiences with streptomycin and dihydrostreptomycin justify the expectation that some of the new antibiotic agents, so rapidly expanding in numbers, will also produce structural damage to body tissues. Whether tissue damage is wholly deleterious, or whether it may offer a new therapeutic approach to disorders of the central nervous system and of the cochlear and vestibular mechanisms must be determined in the case of each new drug.

Since the introduction of antibiotic therapy, the otorhinolaryngologist has revised his concept concerning *the optimum extent of surgical procedures for the control of infectious processes and particularly those involving bony structures.* It has become necessary to regard many time-honored and once standardized, radical surgical procedures in an entirely new light.

Perhaps the most dramatic effect of properly directed antibiotic therapy is observed in osteomyelitis of the skull. During the preantibiotic era, when wide removal of diseased bone was imperative because of the lack of known controls against the spread of infection within bony structures, the operations of Riedel, Killian, Lothrop and Lynch were life-saving measures. Today, they are often no more effective than simpler measures and entail sacrifice of functioning structures and often needless external deformity.

Osteomyelitis, with or without brain abscess, has rarely been cured by antibiotic therapy alone, but antibiotic therapy together with surgical drainage has effected some remarkable cures. Even the view that all necrotic bone must be removed

before healing can occur may need revision. Although the antibiotics exhibit a peculiar ability to relieve the pain associated with osteomyelitis, their main value lies in the prevention of sequestration and in the arrest of progressive extension of infection to contiguous osseous tissue. While their timely use in osteomyelitis, secondary to sinus infection, has not eliminated the necessity for surgical drainage, it has modified greatly the extent and character of effective drainage.

Thus, simple drainage of the frontal sinus, the construction of a permanent epithelial-lined tract between the sinus and the nasal chamber and vigorous antibiotic therapy have been found to be adequate for the complete control of osteomyelitis of the frontal bone.[9] Obviously, more than this will be needed to be done in the presence of subdural or brain abscess, and it is yet to be learned how effective antibiotic therapy will be in osteomyelitic processes extending beyond the frontal bone.

Although immediate sequestrectomy and drainage have been mandatory in the past, it now seems likely that under the influence of antibiotic therapy alone, sequestra may disappear and bone regenerate. The Roentgenologic evidence of this amazing reversal of the pathologic process is supported by the clinical course. Whitfield[12] has reported the effects of combined penicillin and aureomycin therapy in a patient with osteomyelitis of the mandible. The major symptoms appeared one week after the extraction of a molar tooth and consisted of acute cellulitis of the cheek, the cervical and both submental regions and the interior of the oral cavity. Nine teeth were loose and tender. Roentgen examination revealed osteomyelitis of the mandible and early sequestration. Antibiotic therapy, administered as a preoperative precaution, was followed by prompt relief of pain and complete subsidence of the cellulitis on the sixth day. The teeth became firmly set. Neither sequestrectomy nor drainage were required, the patient resumed work on the third day, and all symptoms had disappeared at the end of a month. Roentgen examination four weeks after antibiotic therapy was begun indicated further

resorption of bone, but the sequestrum had completely disappeared. At the end of six weeks bone regeneration was unmistakable.

There are many limitations to antibiotic therapy. Laboratory data and the knowledge gained from basic research can guide the clinician, but the optimal forms of therapy are ultimately determined by clinical trial. In following the course of bone infections, progressive Roentgenologic examinations are essential in assembling the value of any form of therapy. On the basis of Roentgenologic studies on bone infection in the field of otolaryngology, it is becoming increasingly clear that the question of adequate surgical drainage is not "How radical must we be?" but rather "How conservative can we be?"

SUMMARY.

1. Correctly applied antibiotic therapy plays an important rôle in the prevention of residual deafness due to acute otitis media. Irreversible acoustic loss of varying degree results from the indiscriminate use of the antibiotics and the failure to perform paracentesis when it is indicated.

2. The presence of new or unusual microorganisms in the upper respiratory tract is a not uncommon consequence of antibiotic therapy, and they may be associated with bizarre and unfamiliar lesions. In addition to identification of the constituents of the new flora, it is necessary to demonstrate their relation or irrelation to any associated lesions, assess their pathogenicity, and devise therapeutic measures for their control.

3. It is to be anticipated that among the rapidly expanding number of new antibiotics, some will produce toxic effects similar to those of streptomycin and dihydrostreptomycin, and that they will impair the function of cranial nerves other than the VIIIth.

4. The dramatic effects of antibiotic agents in the control of osteomyelitis of the skull have greatly restricted the extent of surgical intervention.

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GRANULAR CELL MYOBLASTOMA OF THE VOCAL CORD.*

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In 1926, Abrikossoff[1] described a new form of striate muscle tumor which has since been designated as granular cell myoblastoma. This tumor is relatively infrequent, the most common site of origin being the tongue. Murphy *et al*,[2] in 1949, collected 219 cases from the literature and added 10 cases of their own. The majority of these tumors are benign; however, a malignant granular cell myoblastoma arising from the urinary bladder[3] and one from the gluteal region[4] have been reported.

There have been but nine cases of granular cell myoblastoma reported as having occurred in the vocal cord and of these, only three have been recorded in the English literature. Besides the report by Klemperer,[5] Iglauer[6] and Fust and Custer,[7] who reported one case each, there was a report in English by Kleinfeld,[8] but his case had been previously reported by Klem perer.[5] Klemperer cited Abrikossoff, Dawydow, and Derman and Golbert as each having reported one case of the larynx. In 1942, Iglauer[6] reported a case and cited Geschelin, Bobbio and Freckner as each having reported one case.

An interesting clinical feature of granular cell myoblastoma, as pointed out by Klemperer, is that there is often a very marked pseudoepitheliomatous hyperplasia of the overlying stratified squamous epithelium that not infrequently results in a mistaken diagnosis of squamous cell carcinoma. There has been no report of malignant granular cell myoblas-

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toma of the larynx. Recurrence after removal is quite common, due to the fact that the edges of the tumor are frequently not well defined and consequently the tumor may be incompletely excised.

The tumors are small, 1 to 2 cm. in diameter, and grossly the lesion appears like a small raised plaque. Its cut surface is light gray or tan in color. The overlying epithelium may appear leucoplacic. At times, there may be a false capsule due to compression of surrounding tissue.

The microscopic picture may be of two different types, namely, the uniform variety and organoid variety.

The uniform variety is composed of sheets and cords of large granular cells which infiltrate insidiously the surrounding tissues. There is a small amount of intercellular tissue stroma. The granular cells are round to oval with eosinophilic granular cytoplasm and small vesicular nuclei. The cell outlines are rather indistinct, and the cells are molded probably by pressure one upon the other. They resemble xanthoma cells but special stains will easily differentiate between the two. Tumors of this variety are considered benign.

The organoid variety is composed of large tumor cells arranged in pseudoalveolar masses separated by very delicate capillaries, so that the tumor suggests the appearance of paraganglioma. The tumor cells have nuclei which are larger and more hyperchromatic than the ordinary uniform variety. The cyptoplasm is acidophilic, finely granular and vacuolated. This organoid variety is considered to be malignant.

Because there have been but three cases of granular cell myoblastoma of the vocal cords previously reported in the English literature, it is believed that the following two cases will be of interest:

CASE REPORTS.

Case 1: The patient, a 63-year-old white male, has been under observation for four years. The patient was of special interest because he had pulmonary tuberculosis prior to being sent to our hospital. Later he did develop laryngeal tuberculosis in the cord opposite to the one having the myoblastoma.

In 1945, he experienced fatigability and severe cough, and raised about 30 cc. of sputum daily. In April of 1946, because of repeated hemoptysis, he was hospitalized and about this time he also became hoarse. For the next two years he was hospitalized in three different hospitals and in each his sputum cultures and smears were positive to acid-fast bacilli; also in each of these hospitals his laryngeal difficulty was diagnosed as tuberculous, but no biopsy was done.

He was admitted to Fitzsimons Army Hospital in September, 1948. The larynx showed a thickening of the left cord with a small nodule near the arytenoid which had a leucoplacic appearance. The rest of the larynx was normal. A removal of the nodule was done by direct laryngoscopy, using basket forceps. The histological diagnosis was granular cell myoblastoma.

The patient had received streptomycin therapy for 112 days in other hospitals, and his sputum remained negative by smear and culture during his entire stay in this hospital. He was discharged from the hospital at his own request in January, 1949.

He was readmitted to this hospital in November, 1949, because of recurrence of hemoptysis and an afternoon fever of 102° F. Indirect examination of the larynx showed thickening of the right vocal cord, arytenoid and in the interarytenoid space. The left cord where the tumor had been removed appeared normal. His sputum remained positive during his entire hospital stay despite continuous tibione therapy. He left the hospital in June, 1950, against medical advice and without completing his course of tibione therapy.

He was readmitted in January, 1952, with the chief complaint of hoarseness, sore throat and chest pain. The right cord and interarytenoid sulcus showed more extensive infiltration and edema; the left cord was normal in appearance. A biopsy from the right cord and interarytenoid mass showed tuberculosis but no evidence of granular cell myoblastoma. At the present time this patient still has considerable infiltration in the right cord and interarytenoid space in spite of an additional 42 days of tibione therapy and six months of streptomycin and para-aminosalycilic acid therapy.

Pathology findings on the tumor removed in October 1948, were as follows: *Gross:* A moderately firm, pearly white, irregular, nodular strip of mucosa measuring 1.2 x 0.3 x 0.2 cm. *Microscopic:* Tissue is covered by stratified squamous epithelium which in places shows a tendency to hyperkeratinization. The basal layer is everywhere intact and shows no evidence of infiltration or anaplasia. In one portion there is a cellular nodule extending from just below the epithelium almost to the base of the section. The nodule is nonencapsulated, poorly defined and merges almost imperceptibly with the areolar tissue surrounding it. Under high power the tumor cells have an acidophilic granular cytoplasm. In general, their cell outlines are indistinct, some cells having an elongated strap-like appearance, while others have a polymorphous appearance (see Figs. 1 and 2).

Case 2: The patient was a 24-year-old colored male admitted to the hospital in March, 1952. He had had hoarseness for two years. When his hoarseness first started he was seen at another hospital and was given a course of penicillin, which improved the hoarseness. In October, 1951, he was again seen at another hospital, but no therapy was instituted. A laryngeal biopsy was taken in February, 1952, at an associate hospital and a histological diagnosis of granular cell myoblastoma was made.

Fig. 1. Case 1. Granular cell myoblastoma of left vocal cord (×100).

When the patient was first seen at this hospital in March, 1952, he had a lesion about 1.5 cm. in diameter, raised with pearly appearing surface, on the left vocal cord near the arytenoid. The patient also had chronic glomerular nephritis and had been treated for syphilis with 63 injections of penicillin in 1946. The tumor was removed by laryngofissure in March, 1952.

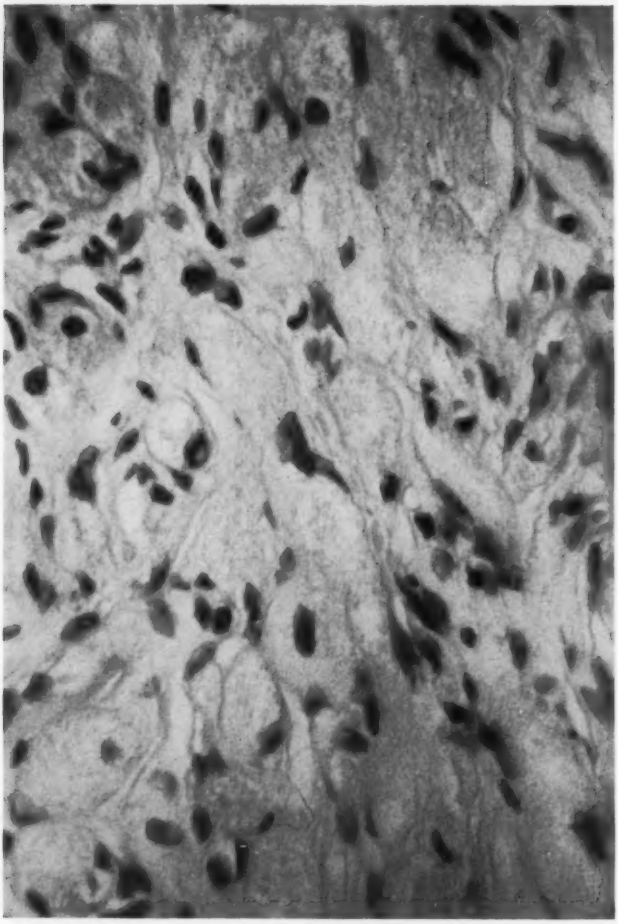

Fig. 2. Case 1. The tumor cells have ill-defined borders and acidophilic granular cytoplasm (×600).

Pathologic Findings: Specimen consists of a plaque of tissue 3 cm. in diameter and 0.4 cm. thick. It consists of a yellowish-gray, firm, membranous structure in the central portion with fragments of muscle surrounding this. Sections show respiratory epithelium covering a loose edematous stroma containing glands. There is mild lymphocytic and

Fig. 3. Case 2. Granular cell myoblastoma of left vocal cord showing the marked pseudoepitheliomatous hyperplasia of the surface epithelium (×100).

plasma cell infiltration in this area. Other fragments show stratified squamous epithelium showing marked hyperplasia and elongation of the rete pegs. Beneath this there are nests of large tumor cells with regular nuclei, and pale, eosinophilic, granular cytoplasm. These cells are adjacent to normal muscle (see Figs. 3 and 4).

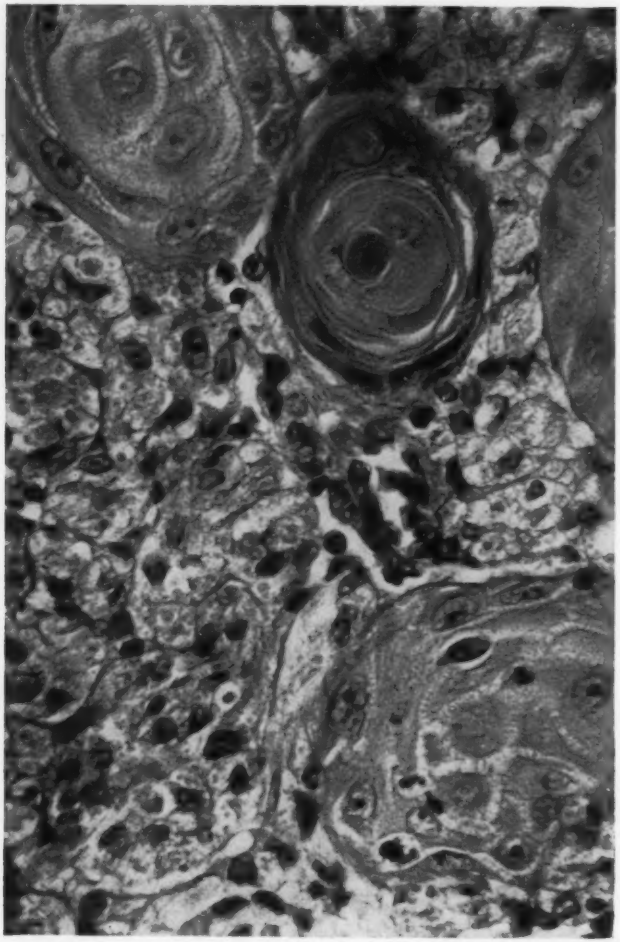

Fig. 4. Case 2. The granular cells surround a nest of squamous epithelial cells (×600).

SUMMARY.

Granular cell myoblastoma of the vocal cord is briefly discussed. Nine cases have been reported previously and only three cases in the English literature. Two additional case reports are presented.

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7. Fust, J. A., and Custer, P. R.: On the Neurogenesis of So-called Granular Cell Myoblastoma. *Amer. Jour. Clin. Path.*, 49:522-535, June, 1949.

8. Kleinfeld, S.: Myoblastoma of the Larynx. *Arch. Otolaryngol.*, 19: 551-555, May, 1934.

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

A sum of money having accrued from the Casselberry Fund of the American Laryngological Association, a prize will be offered in 1954 for original investigation in the art and science of laryngology or rhinology. These must be in the hands of the Secretary, Dr. Harry P. Schenck, 326 South 19th Street, Philadelphia 3, Pa., before March 1, 1954.

ILLINOIS COLLEGE OF MEDICINE COURSE IN BRONCHOESOPHAGOLOGY.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period Oct. 19 through Oct. 31, 1953, under the direction of Dr. Paul H. Holinger.

Interested registrants write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Ill.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

May 1, 1953.

Audicon Models 400, 415, 530 and 615.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Model 11.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3; Dahlberg Model D-4.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Fortiphone Models 20[a] and 21-C.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.

Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Boston, Mass., May 16-17, 1954.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

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Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., Oct. 5-9, 1953.

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Treasurer: Dr. John Louzan.
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Place: Army and Navy Club, Washington, D. C.

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Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

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Meeting: Palmer House, Chicago, Ill., Oct. 16, 1953.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

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Meeting:

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Chairman of Section on Otolaryngology: Leland R. House, M.D.
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Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to June, inclusive—Otolaryngology Section; 6:00 P.M., first Thursday of each month from September to June, inclusive—Ophthalmology Section.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

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Secretary General: Dr. Victorino Marquez Reveron.
Secretary of Assemblies: Dr. Cesar Rodriquez.
Time and Place: Caracas, Venezuela, July 31, 1954.

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Meeting, Joint: Charleston, S. Car., Sept. 13-16, 1953.

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President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ontario.
Time: June 14-18, 1953.

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Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: Second International Congress of Bronchoesophagology.
Time and Place: June 15 and 16, 1953, Albert Plage, Le Zoute, Belgium.

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